THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—13TH YEAR. SYDNEY: SATURDAY, OCTOBER 16, 1926. No. 16.

Table of Contents

THE NEED FOR PROPAGANDA BY THE MEDICAL PROFESSION WITH REGARD TO CANCER.[1]

By SIR GEORGE SYME, K.B.E., M.S. (Melbourne), F.R.C.S. (England), F.A.C.S.,
Melbourne.

IN Moliére's drama *"Le Médicin Malgré Lui,"* the medical profession is satirized and ridiculed and it is suggested that the whole duty of physicians consists "in pouring drugs of which they know little, into bodies of which they know less." While such a view of the profession is absolutely unjustifiable today, it may be questioned whether the general body of its members realizes even now the full extent of its responsibilities and functions. In the presidential address to the last Medical Congress I endeavoured to refute some accusations by the press that the medical profession was indifferent to the prevention of disease, but I had to admit that there was some truth in them. The profession realizes that prevention of disease is the duty of some of its members, of specialists who devote their whole time to it as officials. It is true, however, and it is regrettable that most practitioners restrict their activities almost entirely to endeavouring to cure existing diseases and do comparatively little to prevent its onset or to investigate the causes or nature of the diseases they see in practice. They say they have no time and probably say so truly in some cases. But are not their omissions in these respects due not so much to want of time as to want of thought and want of interest? Has it ever occurred to them that they could or ought to do anything in the way of investigation or prevention? The General Medical Council of Great Britain by a resolution in regard to medical education which came into force on January 1, 1923, determined that "throughout the whole period of study the attention of the student should be directed by his teachers to the importance of the preventive aspects of medicine." If this resolution be faithfully carried out, the next generation of practitioners will not fail from want of knowledge, but may still do so from want of interest, owing to what Osler called "that accursed apathy, the chief foe the medical profession has to fight."

I would remind you that one of the chief arguments against "nationalization of medicine" is that it would tend to destroy the peculiar, intimate and

[1] Read at a meeting of the Victorian Branch of the British Medical Association on September 1, 1926.

confidential relations that should exist between practitioner and patient. But do we recognize the full implications and responsibilities of this relationship? To what extent do medical practitioners make use of it to carry on propaganda with regard to the prevention of disease? In this connexion I would like to quote a passage from an address given by Lord Dawson to the Thirteenth Convocation of the American College of Surgeons, last October, when he said:

> Teaching will become a prescribed duty in the doctor's career. How can it be otherwise? If we are to get to the beginning, if we are to guide people in the ways of health, if the community is to guard the health of its mothers, its babies, its school children, its industrial workers, the family doctor must become an educationalist and in part a health administrator. If he does not, his rôle will suffer progressive diminution, curtailed as it will be on the one hand by the whole time health official and on the other hand by the invading specialist. The family doctor should remain the foundation of medical service, but his outlook, functions and training need modification to meet changing needs. First must come his care of the sick, but beyond that he will have communal and educational duties. Withal let us do nothing which would impair the personal touch, the deep and abiding interest which means so much in the house of sickness, for our rosary needs to be strung with the beads of love as well as with those of thought.

The founders of the British Medical Association expressed the opinion at their inaugural meeting at Worcester in 1832 that their chief object was "to raise admiration of the medical art in the public mind." Surely that object must include measures disinterestedly carried out by the members, collectively and individually for the public benefit, such as propaganda with regard to prevention of disease. Yet, when it was proposed in the Council of the Branch to establish a cancer propaganda committee, several members strongly objected and said the proposal was *ultra vires* and not desirable. After a favourable legal opinion had been obtained the proposals were eventually carried. The Cancer Committee has held meetings and has prepared various methods of informing the public on the subject, such as wall sheets, addresses to be broadcast, pamphlets, articles in the press, lectures and so on. The Committee now appeals to members of the Branch to assist in the work of propaganda. I suggest that they could help most materially by discussing various aspects of cancer with their patients in the course of that peculiar relationship which should exist between them. People will pay much more attention to what they are told in a personal way by a trusted adviser than to what they read on a wall sheet for instance or hear when "listening in" and medical advisers could also distribute literature on the subject, prepared by the Committee and have it in their waiting rooms. Instruction could also be given at health centres, like that at Prahran or at baby health centres. Medical practitioners could in this way contradict erroneous popular ideas about cancer. For instance, it is very generally believed that nothing is known about cancer. Patients could be personally told that while we do not know very much about the actual cause of cancer, we do know a great deal about its nature and mode of spread in an affected individual, knowledge which is of great value in its treatment. It is also a prevalent impression that cancer is inherited and this often causes unnecessary alarm in people's minds. They can be told that the inheritance of cancer has not been proved and is not generally accepted by the profession. Above all should it be explained that cancer must not be regarded as a hopeless and incurable condition, but that on the contrary, if it is treated sufficiently early, approved modern methods give great hope of cure. In an address on this subject, given last December, Dr. D. C. L. FitzWilliam said:

> Accessible cancer should yield a cure in 80% to 90% of cases and such figures had actually been obtained in early cases in the breast and tongue, where otherwise the ordinary mortality figures were as bad as anywhere in the body.

But it is essential that cancer should be recognized in the earliest possible stages. To get such cases early practitioners should teach their patients what the indications of these early stages are. To do so it is necessary that every medical practitioner must be thoroughly familiar with these indications. Unfortunately it is to be feared that a proportion of the medical profession does not know them or fails to recognize them, perhaps because many textbooks still describe the advanced conditions of cancer. The diagnosis of the early stages may be difficult, but it is the bounden duty of all practitioners to make themselves familiar with the early symptoms and signs and with possible precancerous conditions. They should always have the possibility of malignant disease in mind, be on guard and suspicious and get expert assistance early. A frequent mistake that has come under my own observation, is to regard an early carcinoma in the breast as a mastitis, especially when in a lactating breast. The diagnosis may be difficult even for an expert and in my opinion the safest course is to remove the breast as soon as possible and submit it to microscopical examination. If found malignant, more extensive operation should follow. If non-malignant, no harm is done, as the mastitis hardly ever recovers completely and the breast is left permanently damaged. Another organ in which carcinoma is often overlooked in the early stage is the uterus and generally because the practitioner does not make a sufficiently thorough examination or cooperate with the pathologist. It is not sufficient to curette the uterus and send some indefinite scrapings without any information to the microscopist. An erroneous report may be sent back because the scrapings are insufficient and do not include any of the malignant tumour, although it may have been present in the uterus. In the tongue cancer often developes on a syphilitic base and when the malignant change begins, the practitioner may consider it still syphilitic and continue medical treatment too long. Every sore on the tongue should be excised and submitted to microscopical examination even in a syphilitic patient, if it does not promptly yield to specific treatment.

Want of thorough examination may be responsible for overlooking early cancer in the rectum. This condition is not infrequently associated with piles which may be regarded as the only trouble.

In all conditions which a patient calls piles, the whole rectum should be carefully examined. The early symptoms are often obscure and referred to the abdomen and in any abdominal condition it is wise to make a thorough rectal examination. Mistakes are more excusably made in not recognizing early cases of cancer in other parts of the alimentary canal, but here also the practitioner should be alert as to the possibilities and prompt to get expert assistance, such as chemical examination of stomach contents and fæces, use of œsophagoscope, X rays *et cetera*. These will often reveal enough to justify surgical exploration. In my experience patients are usually quite willing to undergo exploratory operations in such cases and think them well worth while if the operation proves that no malignant disease is present.

While early operations are satisfactory, it may not be out of place to deprecate extensive operations in very advanced cases. I believe that it is the bad results of late operations that are largely responsible for the popular impression that cancer is incurable. Of course, it must always be a matter of personal judgement in a particular case as to whether it is too late for a satisfactory result, but it is common knowledge that some surgeons advocate operations in very advanced cases. It must also be a matter of personal judgement whether in some cases, especially about the mouth and pharynx, the penis and others, diathermy combined with radiotherapy may not be preferable to extensive cutting operations. These observations, however, are rather by the way. What I want to urge is the need in this country for propaganda on the subject of cancer.

The Minister for Health in the Commonwealth recently pointed out that the death rate from cancer is higher in Australia that in seventeen other countries. In the journal *Health* for May, 1926, Dr. M. J. Holmes has analysed some cancer statistics and asserts that in recent years there has been a very pronounced progressive and serious increase in the mortality from cancer in Australia. In 1870 it was 26 per 100,000 of mean population; in 1900 it was 63 per 100,000 and in 1923 it was 89 per 100,000. It was estimated that if the mortality continued to increase as it had done, the number of persons now living in Australia who would die of cancer, approached 750,000 or one in eight of those now living and in fifty years the ratio would be one in five.

In America a great deal of propaganda work with regard to cancer has been carried out by the American Society for the Control of Cancer with very good results. In illustration the following quotations may be given from a publication by the society, entitled: "How Cancer Education Pays." This is a review of the last report of the Pennsylvania Cancer Commission. The findings of the Commission are based on a study made in 1910 and repeated in 1923 under circumstances which make the data collected for the two years comparable. The review says:

> Thirteen years of education have cut down the average time between the discovery of the first symptom in superficial cancer and the first call on the doctor from eighteen months to 14·6 months or 20%. In cases of deep-seated cancer the interval has been reduced nearly one-half. The doctors of Pennsylvania have learned the importance of prompt action sufficiently to have reduced the interval between the patient's first appearance and the institution of the treatment required from thirteen months to 4·5 months or 65% in superficial cancer; and from twelve months to 3·9 months, or about 70% in deep-seated cancer. . . . Most of the members of the medical profession are now serving the community well in instituting early treatment for cancer, but there still remain about 10% whose attitude is far from right. . . . In many instances where people have applied unusually early the reason given is "heard a lecture on cancer." The reports show that in general it is the younger patients, especially the younger women who apply for radical treatment soonest. We believe this is a direct result of the educational campaign. The Commission believes that this report proves conclusively that cancer education pays.

The Health Association of Australasia has done some propaganda and has issued a useful wall sheet. But more is necessary. Will the members of this Branch assist in carrying on education on cancer by every possible means?

RECENT RESEARCHES ON CANCER.[1]

By C. H. Kellaway, M.D., M.S. (Melbourne), M.R.C.P. (London),
Melbourne.

Despite numerous clinical, statistical and experimental studies of the cancer problem, we are still ignorant of the exact cause of the disease and can only speculate in regard to the possibility of its cure by other means than are afforded by surgery or the use of X rays and radium. This is not to say that we know only a little about cancer. On the contrary a great many facts have been established concerning it.

In spite of its wide distribution cancer is specific for every species and transplantation can be carried out only from individual to individual within the same species. When transplantation is successful, the new growth which forms, does so by the multiplication of the introduced cells except in a few cases; in these the reacting tissues of the host themselves become malignant.

Cancer is at first strictly localized, but when spread takes place by infiltration, permeation or metastasis, the cells which are distant from the place of origin, retain their biological and structural characters. The cancer cell has the capacity of uncontrolled growth. It is practically independent of the nutrition of the rest of the body, though the growth of some transplanted cancers is said to be affected by the presence of excess of vitamins or of cholesterol in the diet. Warburg has shown that cancer cells, unlike rapidly-growing embryonic cells, do not show any increase in the rate of respiration over that of normal tissues, even when nutritive substances are present in abundance. Under anaerobic conditions *in vitro* they can convert sugar to lactic acid and lose this power only

[1] Read at a meeting of the Victorian Branch of the British Medical Association on September 1, 1926.

when they have lost their vitality and can no longer be successfully transplanted. Glycolysis still goes on when the tumour cells are placed under aerobic conditions. In this also they differ from normal cells. In the case of muscle, for example, glycolysis and oxidation both occur, but oxygen consumption causes the lactic acid to disappear, whereas there is little difference in the amount of lactic acid produced by cancer cells under anaerobic or aerobic conditions. In cancer an abnormal type of metabolism has been developed and the splitting of carbohydrate to lactic acid provides the energy of the growth process.

The difference between benign and malignant growths in this respect is only one of degree. Papillomata like carcinomata exhibit anaerobic glycolysis, but the energy provided by oxidation is much greater in proportion in benign tumours. Embryonic tissues form lactic acid in large amounts under anaerobic conditions, but when oxygen is present this glycolytic activity is masked and respiration causes the products of glycolysis to disappear. If embryonic tissue is kept at body temperature for some hours under anaerobic conditions in the presence of an excess of glucose, when oxygen is admitted oxidation does not occur, though glycolysis goes on. Warburg has suggested that deficient oxygen supply brought about by inflammatory or other processes may be the cause of cancer, those cells with most glycolytic power surviving and multiplying.

Often repeated or chronic irritation frequently gives rise to cancer and when it does so there is usually a long latent interval. There are a good many substances which can function as specific irritants. Cancers may be readily produced, for example, by repeatedly tarring small areas in laboratory animals. In this country cancers which can definitely be attributed to specific irritation are of infrequent occurrence.

Many interesting facts are accumulating in regard to the production of susceptibility and immunity to cancer. For some years it has been recognized that the injection of embryonic extracts into animals leads to increased sensitiveness to the growth of transplanted tumours (Bashford, Murray and Haaland) and that such extracts also increase the rate of growth of tumour tissues *in vitro* (Carrel and Drew). More recently Chambers and Scott have shown that if tumour tissue is emulsified, repeatedly washed with saline solution and allowed to autolyse at 42° C. in a solution saturated with toluene, a clear colourless fluid is obtained which contains but little protein, but which when inoculated into animals makes them more susceptible to tumour growth as tested by the subsequent inoculation of a transplantable tumour. This stimulating substance appears soon after the onset of autolysis and persists for several days. A stimulating substance can also be produced by autolysis of tumour substance in one-twentieth normal hydrochloric acid. It appears to withstand heating to 100° C. for ten minutes. According to these authors it is therefore a chemical substance rather than a living agent. Carrel and Baker have recently reported that growth-stimulating substances are present in Witte peptone and also among the products of digestion of proteins such as fibrin and egg albumin. They prepared a solution which was considerably more potent than the original digest by precipitating the coagulable proteins with tri-chlor-acetic acid and by subsequent purification of the proteose fraction by repeated precipitation with saturated sodium sulphate and dialysis. In this connexion it is interesting to note that according to Cramer tar cancers are more rapidly produced in animals in which, after removal of the spleen, the minced splenic pulp has been introduced into the peritoneal cavity.

Immunity may also be produced against experimental cancers. This may be demonstrated in the case of tar cancer by the production and surgical removal of a growth after which the animal appears to be insusceptible to subsequent tarring or to the transplantation of cancer. The inoculation of irradiated tumour material is also said to cause some degree of immunity. The experiments of Lumsden with tumour antisera are also suggestive. These antisera are potent *in vitro* and in the living animal act most powerfully when they are injected locally and prevented from escaping into the general circulation by constriction of the vascular supply of the tumour. In animals with two transplanted tumours cure of the one is followed by retrogression of the other. Recent work by Itami with transplantable tumours of mice has shown that resistance to tumour transplantation can be brought about by previous treatment with extracts of normal tissues. Injection with whole blood, with washed leucocytes, with tissue from lymphatic glands or spleen, lung, skin or heart muscle gives rise to a definite degree of immunity.

About a year ago Gye put forward the theory that cancer is a specific disease of the body cells caused by a virus or by a group of viruses but in which infection is determined by the presence in the cells of a chemical factor with a strict species specificity. The experimental evidence upon which he based this view was briefly as follows: Rous in 1911 had described a sarcoma of plymouth rock chickens which could be propagated by the injection of dry powdered tumour or of filtered extracts, these latter losing their power to cause new growth if treated with chloroform. Gye found that when a piece of this tumour was placed in an appropriate culture medium and incubated anærobically the supernatant fluid retained its power of producing tumours for some days. After more prolonged incubation or repeated subculture in a medium containing a fragment of chick embryo it yielded "cultures" which were not infective. A filtrate of the original extract which was active in very small doses was deprived of its power of producing tumours even in much larger doses by treating it with chloroform. This chloroform filtrate when injected into plymouth rock chickens, together with subcultures of the Rous tumour, of cultures which had become inactive after prolonged incubation or

of cultures of mammalian tumours, was once more able to cause the growth of the sarcoma of the fowl. Gye showed also that by centrifuging at very high speed "primary cultures" of Rous sarcoma (which had been incubated only for a short time) the top layers became much less potent in producing new growth while the deposit remained active. This deposit lost its activity after repeated washing, but regained it when some of the supernatant fluid was added. Gye interpreted these results as showing that two factors were present in the original tumour extracts, a particulate virus capable of separation by centrifugation at high speed and of multiplication under the conditions of culture and a chemical factor gradually destroyed by prolonged culture.

The weakest link in this chain of evidence lies in the inactivation of the Rous virus by chloroforming the extract. Murphy has recently claimed to have reactivated chloroformed filtrate with cultures containing instead of tumour chick embryo and rat placenta which share with malignant tissue the capacity to utilize sugar in the absence of oxygen. He suggests that the causative agent in the production of the chicken tumour may be an enzyme-like substance which is inactivated by chloroform and reactivated by a diffusable substance from malignant tumours, embryonic and placental tissues. Alternatively the chloroform may simply attenuate the causative agent to a point at which unaided it is too weak to cause tumour formation, but can do so in association with some injurious or stimulating product of the metabolic activity of tissues grown under these anærobic conditions. Murphy points out that the fact that a large excess of chloroform apparently destroys the active agent so that no reactivation is possible is in favour of the latter view. Kolmer and his associates, using varying amounts of chloroform for inactivation of the filtered extract of Rous sarcoma, have been able only to cause tumours with filtrates by the addition of cultures when the inactivated filtrate was itself capable of causing tumour formation when injected alone. Harde has also drawn attention to the importance of change in hydrogen ion concentration of extracts, a slight increase in acidity greatly increasing their growth-producing powers. He explains in this way Gye's experiments in which a supposedly particulate virus was thrown down by centrifugation of extracts, the deposit after washing being reactivated by the acid supernatant fluid.

There remains, therefore, no clear evidence of the participation of a virus or group of viruses in the ætiology of cancer. The influence of chronic irritation in causing experimental cancer, the strict species specificity necessary for successful transplantation, the stimulating action of products of autolysis seem to point rather to the occurrence of some such process as Warburg has suggested, resulting in the local evolution of a race of cells with a dominating anærobic metabolism whose growth is stimulated by the autolytic products, derived from those normal cells which fail to survive.

On the therapeutic side the work of Blair Bell and his colleagues at Liverpool has aroused much interest. Using colloidal suspensions of metallic lead prepared electrically, they have treated by intravenous injection a series of two hundred and twenty-seven patients suffering from cancer, in some forty of whom the results appear to be satisfactory. It is not possible at this early stage to estimate the value of this treatment but it is by no means improbable that some more efficient and less dangerous method may be evolved by work carried out along these lines.

Despite the hopes which have been aroused of an early solution of the cancer problem this end does not appear to be in sight and it behoves us to attempt to lessen the appalling mortality of the disease by means which lie at our hand.

Even if the cause of cancer were known and cure were certain, there would still remain the necessity for education of the public so that the cancer patient might be subjected to treatment in the early stages of his malady. Both now and in the future propaganda will be a necessary weapon in the campaign against cancer.

The Cancer Propaganda Committee of this branch of the British Medical Association has been formed with a view to providing authoritative information for use in the educative campaign of such bodies as the Health Association of Australasia which has been and is actively engaged in such work. Your Committee has edited a booklet for the Colonial Mutual Life Assurance Society which will have a wide circulation and has prepared wall sheets, lectures for broadcasting and press articles.

To be effective such propaganda work requires the active cooperation of the medical profession and it should be recognized that the education of the public in regard to those symptoms which may indicate the presence of a cancer in its early operable stage is a moral obligation upon every medical man.

LUMBAR PUNCTURES AND ACIDOSIS IN EPILEPSY AND ALLIED CONVULSIVE DISORDERS.

By Guy P. U. Prior, M.R.C.S. (England), L.R.C.P. (London),
Medical Superintendent, Mental Hospital, Parramatta;

and

Alfred T. Edwards, M.B., D.P. (Sydney),
Medical Officer, Mental Hospital, Kenmore, Honorary Psychiatrist, Goulburn Hospital.

We would venture to point out the great usefulness of lumbar puncture as a therapeutic agent in the treatment of some mental disorders, more especially in cases of *status epilepticus* and allied conditions.

We present notes of fifty-three patients upon whom we have done this operation. These are only a fraction of the total, as for many years we have performed lumbar puncture as a routine treatment in all cases of *status epilepticus* and on general

paralytics in convulsions. It was Dr. Oliver Latham who first pointed out to us the value of the withdrawal of cerebro-spinal fluid in this class of case.

We have divided the conditions under the following headings: *Status epilepticus*, epileptiform convulsions of general paralysis, epileptic equivalents, chronic mania (as sedative), convulsions with arteriosclerosis. We have also carried out the operation in epilepsy as preventive of a fit.

Status Epilepticus.

In cases of *status epilepticus* or serial epilepsy after the withdrawal of from twenty to sixty cubic centimetres of cerebro-spinal fluid the benefit is often immediate and complete. It is our custom in treating these conditions to give first a soap and water enema and, if this fails, to give an enema of potassium bromide eight grammes (two drachms) with chloral hydrate 1·2 gramme (twenty grains). These in less severe cases will often cause a cessation of the attacks, in fact it is surprising how often no other treatment than the soap and water enema will bring about the desired result. We are of the opinion that nothing is of any use until this simple measure has been taken and the relief often obtained would almost suggest that an intestinal stasis plays some part in the causation of *status epilepticus* or the result may be due to an alteration in the blood pressure and so in the cerebro-spinal fluid pressure. It is desirable to drain thoroughly the cerebro-spinal fluid, no matter what quantity be obtained. When the flow is synchronous with the respiration, the fluid is at a sufficiently low pressure.

The results of the punctures for serial epilepsy or *status epilepticus*, are given in Table I.

A glance at this table shows that in the majority of cases the attacks cease or are greatly lessened almost at once. Four patients died, all of these *post mortem* manifesting some gross brain lesion.

Case 13 was one of giant celled glioma. In Case 14 a cortical tumour was present the tumour being in the parietal lobe, just behind, pressing upon but not involving the Rolandic convolution. Case 15 was one of cortical hæmorrhage in an epileptic of long standing. In Case 27 no *post mortem* examination was made, but it was a case of traumatic epilepsy, the injury having left the patient hemiplegic.

Of the three cases recorded as being followed by no benefit that could be attributed to the puncture, two were of traumatic origin. Both patients had been trephined, in Case 20 several operations having been performed. One case, 16, seemed to be only of idiopathic epilepsy in an imbecile; in two series the patient did not respond to lumbar puncture.

In Cases 8 and 17 recovery occurred only after "intensive treatment." In Case 8 the patient was a child of thirteen years who on two previous occasions had attacks in long series. These left her extremely ill with such absolute incoordination of muscular movement that she could neither walk nor stand alone and had a tendency to fall backwards. On this occasion the first puncture

Table I.

Number.	Age.	Pressure.	Amount in Cubic Centimetres.	Condition Before Puncture.	Result and Remarks.
1	37	+++	20	8 fits in 2 hours	None next four days.
2	71	+++	33	10 fits in 53 minutes	Two during next four days.
3	36	++++	40	6 fits in 30 minutes	None during next week.
4	22	++	18	4 fits in 15 minutes	None during next week; acetone in cerebro-spinal fluid.
5	55	+	23	*status epilepticus* for 60 min.	Four during next three days.
6	36	++	40	*status epilepticus* for 60 mins.	No fits during next two weeks.
7	12	+++	28	*status epilepticus* for 120 min.	Seven next three days; acetone in cerebro-spinal fluid.
8	13	++	20	19 fits in 6 hours	Recovered after extensive treatment; acetone in cerebro-spinal fluid and blood.
9	22	+++++	110	*status epilepticus* for 3 hours	Two fits next seven days.
9	22	++	25	*status epilepticus* for 1 hour..	One during next seven days.
10	13	+++	40	13 fits in 66 minutes	Six during next four days; acetone in cerebro-spinal fluid.
11	18	+++	30	20 fits in 60 minutes	Two fits following two days; acetone in cerebro-spinal fluid.
12	19	++	30	Monthly average of 65 fits ..	Sixteen following month.
12	19	++	40	5 daily for 3 months	None next five days.
13	22	++	30	*status epilepticus* for 60 mins.	*Obiit. Post mortem* examination revealed giant celled glioma; acetone in cerebro-spinal fluid and blood.
14	27	++	30	*status epilepticus* for 60 mins.	*Obiit. Post mortem* examination revealed cortical tumour; acetone in cerebro-spinal fluid.
15	45	−−	2	*status epilepticus* for 2 hours	*Obiit. Post mortem* examination revealed cortical hæmorrhage.
16	18	++	30	Fits for 24 hours	No immediate benefit.
16	18	++	20	Fits for 36 hours	No immediate benefit.
17	55	+++	50	*status epilepticus* 12 hours—extremely ill	Recovered after extensive treatment.
18	50	++	30	*status epilepticus* 1 hour ..	None during next week.
19	37	+++	35	*status epilepticus* 1 hour ..	None during next week.
20	20	+	25	Would have as many as 2,000 fits in about 2 weeks	No benefit from puncture; epileptic of traumatic origin.
21	32	++	35	*status epilepticus* for 2 hours..	None during next week.
22	36	+++	24	40 fits in continuous series ..	None during next week.
23	37	+	10	17 fits in 45 minutes	None during next week.
24	26	2	16	3 fits in 30 minutes	One fit immediately after puncture.
25	45	++	25	30 fits in 6 hours	Sixteen fits twelve hours; *Obiit.* Cerebral tumour.
26	23	++1	56	90 fits between 8 a.m. and 1 p.m.	Acetone—none in blood or cerebro-spinal fluid on tenth; two days later in blood.
27	47	+++	30	*status epilepticus* for 1 hour..	Acetone in cerebro-spinal fluid and blood; epileptic of traumatic origin; *Obiit* shortly after puncture.

reduced her fits from nineteen in six hours, to ten in the next twenty-four hours, but she continued ill with intermittent attacks of increasing frequency and was only semiconscious. Three days later the urine was found to contain a trace of sugar, acetone and diacetic acid. She was again submitted to lumbar puncture and was bled three hundred and sixty cubic centimetres (twelve ounces). Both the blood and cerebro-spinal fluid now contained acetone, which the fluid from the first puncture did not show. She had developed a condition of extreme hypotonia of all muscles, including face, neck and body and her breathing became almost imperceptible, indicating also hypotonia of the diaphragm. Associated with this was an extensor plantar reflex. From this hypotonia the *quadriceps femoris* was exempt and the knee jerks were exaggerated. All other tendon reflexes were abolished as well as the superficial reflexes. The pupillary reflexes were present. The acidosis was treated by giving one hundred grammes of glucose by the mouth. A pint of saline solution was introduced into the cellular tissue and she was ordered bicarbonate of soda four grammes (one drachm) every hour. With the disappearance of the acid bodies she made a quick recovery.

In Case 17 the patient was in a condition of *status epilepticus* from October 30 to November 2. He was submitted to lumbar puncture on November 1 with no benefit and all other known treatment was tried, including hyoscine hydrobromide with no better results. On the morning of November 2 he was very ill, breathing rapidly and was very cyanosed. He was in fact apparently *in articulo mortis*. He was bled five hundred and sixty cubic centimetres (twenty ounces) and an equal amount of saline solution together with 0·5 cubic centimetre adrenalin was administered intravenously. After this he had two attacks and was conscious within six hours.

Case 9 is noteworthy because of the large amount of fluid withdrawn, namely one hundred and ten cubic centimetres, this being most unusual. The patient had had epilepsy from infancy and was hydrocephalic. Whilst the fluid was flowing she had a convulsion, whereupon the stream was broken into little jets, synchronous with clonic muscular contractions, indicating the intimate relationship between cerebro-spinal fluid pressure and blood pressure.

Preventive Puncture.

In two cases of epilepsy we have done the puncture as a preventive measure.

One was a case of myoclonic epilepsy. The patient suffered from coarse tremors for twenty-four hours or more before an attack and we performed lumbar puncture when the patient was in the tremors. This patient had an extremely severe fit immediately after the puncture. In this instance the discomfort and upset created by the operation probably hastened the attack.

One of us (A.E.) conceived the idea of doing lumbar punctures as a routine treatment for severe epilepsy and this was adopted in Case 12.

For years this patient had been deteriorating in spite of treatment. In June, 1925, his fits were reduced by puncture from sixteen in twenty-four hours to none during the following week. The benefit obtained was overlooked and for six months before a second puncture he averaged sixty-three fits a month. After the second puncture the fits ceased for four days, followed by two fits on three successive days. Puncture was repeated and during the next month he had sixteen fits. At the end of this month he was again submitted to lumbar puncture with a further reduction to twelve for that month. Previous to the second puncture the patient was dull, hypotonic, inert, suffered from anorexia and was losing weight rapidly. He became lively, a keen eater, interested in his environment and so mischievous as to be a nuisance to other patients.

General Paralysis of the Insane.

For general paralytics in a condition of extreme restlessness, in succession of convulsions or localized or general muscular twitching which so often occurs in these affections, we have found withdrawal of cerebro-spinal fluid very beneficial and the general condition of the patients has greatly improved for a time. We have notes of nine such patients, all of whom made temporary improvement. These are shown in Table II.

Table II.

Number.	Condition Before Puncture.	Result.	Remarks.
28	General muscular spasms for 36 hours ..	Ceased 2 hours	Pressure ++.
29	General convulsions for 12 hours	Ceased.	
30	Convulsions for 8 hours	Regained consciousness and convulsions ceased one hour after puncture	
31	Extreme restlessness	Slept for 4 hours.	
32	Convulsions for 12 hours	Ceased	Pressure ++++; acetone present.
33	Convulsions for 6 hours	Ceased, slept 2 hours and then regained consciousness.	
34	Suddenly paraplegic with slight general muscular spasms	Spasms ceased almost immediately ..	Pressure ++; amount 22 cubic centimetres; cells 6; albumin +.
35	Twitching of face for 36 hours	Ceased 12 hours after puncture, general condition improved	Amount 10 cubic centimetres.
36	Twitching left side of face and right limbs and other generalized for 48 hours	Ceased in 3 hours	Amount 15 cubic centimetres.

Table III.

Number.	Condition Before Puncture.	Condition After Puncture.	Remarks.
37	Very restless and sleepless for a week	Immediate improvement.	
38	Noisy and restless 3 days	Quiet after	29 cubic centimetres removed; pressure ++; severe headache.
39	Noisy and restless 7 days	Immediate improvement ..	10 cubic centimetres removed; pressure normal.
40	Acute maniacal 3 days	Quiet within 2 days	40 cubic centimetres removed; pressure ++++.
41	Maniacal 7 days	Improved and quiet within 12 hours	Pressure +.
42	Acute epileptic excitement 4 days ..	Recovered within 24 hours..	Removed 30 cubic centimetres.

Epileptic Equivalents.

Those taken as epileptic equivalents are cases from 37 to 42 inclusive. The patients were epileptics who, although having no fits at the time, were in a condition of great restlessness or who were quite maniacal. All of these improved for a time.

In cases 38 and 39 are two of those patients who have but few fits, but who often go through maniacal attacks; in Case 39 the attacks generally lasted for two or three weeks.

In Cases 37 and 42 the patients died a few months later from their epileptic condition. In Cases 40 and 41 the patients have had no return of their acute maniacal condition.

These patients are shown in Table III.

Chronic Mania.

In Cases 43 to 50 we have done this operation for patients suffering from chronic mania who for long periods have been noisy both night and day, sleepless, restless and aggressive and upon whom neither sedatives, drugs nor baths have had effect. In each case the fluid has been under increased pressure and the puncture has acted as a sedative. After the puncture the patients have slept for some hours and have been quieter and calm for from a few hours to seven or ten days. Two of these patients suffered from severe and long continued headache as an after-effect of the puncture.

Although the results of removing the fluid in the cases of chronic mania have given temporary benefit, we have hesitated in extending the treatment in this class of patient, because of the impossibility of being assured that the patient will rest after the removal of the fluid.

Arteriosclerosis.

In Cases 51, 52 and 53 the patients were suffering from epileptiform convulsions, associated with arteriosclerosis.

In each case the convulsions had commenced after fifty years of age and probably when the arterial change was advanced. All three failed to react to the Wassermann test both in the blood and cerebro-spinal fluid. One was a middle-aged man whose blood pressure was consistently over 200 millimetres of mercury. He at irregular intervals would suffer from extremely severe and long continued convulsions, which would apparently bring him to the brink of death. The attacks would cease immediately after drawing off the fluid which was always under increased pressure, and generally about sixty cubic centimetres would be removed. By this means he was saved from death eight times in three years.

In case 53 the patient was a man of sixty years of age with a blood pressure of 230 millimetres of mercury who was in one continuous convulsion for twenty minutes. His convulsion ceased and he regained consciousness within five minutes of the puncture.

In the other case, Number 52, the patient was a woman with an equally high pressure and in her case the treatment was equally satisfactory.

Acetone in the Cerebro-Spinal Fluid.

In sixteen cases the cerebro-spinal fluid has been examined for acetone, the method used being that recommended by Joslin for testing blood for acetone. In nine cases of the sixteen the fluid gave a positive result. Eight of these were epileptics in a condition of *status epilepticus* and one was a general paralytic suffering from severe convulsions. The fluids giving a normal result were taken from the same class of patients, except two who were primary dements and these were taken as controls, and two were suffering from arteriosclerosis and had convulsions at the time the fluid was taken. If the depth of colour obtained can be taken as an indication of the amount of acetone present, most of these must have contained quite a large amount. We are not aware if acetone in the cerebro-spinal fluid has been previously observed in cases of *status epilepticus* or in other forms of convulsions, nor can we offer an acceptable explanation for its presence. In four of these patients the blood was also examined, that is in Cases 8, 13, 26 and 27. In all four the blood was found to contain acetone. In Case 27 the patient died after two hours of *status epilepticus* and very shortly after the puncture; his blood as well as the fluid contained a large amount of acetone.

Case 25 was one of a lad who for ten days had intermittent series of fits. He was bled five hundred and sixty cubic centimetres (twenty ounces) on November 10 and had previously been submitted to lumbar puncture, but neither cerebro-spinal fluid nor blood then contained acetone. Three days later, as his fits still continued, he was bled another three hundred and sixty cubic centimetres (twelve ounces) and this time his blood contained acetone.

Case 8, already described, was somewhat similar, the acetone being detected in the cerebro-spinal fluid, blood and urine, after the patient had been taking the attacks for some days and not early in her illness.

In Case 13 the patient did not respond to treatment, but died after two days of *status epilepticus*. Her blood and fluid contained a considerable amount of acetone. After twenty-four hours' illness the urine contained acetone, had an acidity of 62, ammonium nitrogen of 0·0448 gramme *per centum* and amino acid nitrogen of 199·8 milligrammes *per centum*. *Post mortem* this patient was found to have had a giant-celled glioma of left occipital lobe. She also presented the other *post mortem* signs so often found in epileptics, signs such as a small aorta, persistent thymus and degenerate liver.

In Case 54 the patient, a girl, aged twenty-two years, was an epileptic since infancy. She had been dull for two or three days with furred tongue, but had had no fit for several months. On February 17 about 2 p.m. she collapsed and became suddenly very ill, having in the forenoon been about and worked as usual. When seen her pulse was 130 per minute and respiration 100 per minute. The urine contained much acetone, its acidity was 65, ammonia nitrogen was 0·1496 gramme *per centum*, urea nitrogen was 2 grammes *per centum*. The blood contained acetone, the cerebro-spinal fluid was not examined. The pulse and respiration remained rapid, the respiration nearly as rapid as the pulse and she died after twenty-three hours' acute illness and no abnormal physical signs, except those pointing to an acidosis, were detected. No *post mortem* examination was allowed.

The acidosis in this last case cannot be directly connected with her epilepsy, but shortly afterwards we had another epileptic girl who had a prolonged pyrexia, a highly acid urine containing acetone and diacetic acid, but not sugar. This was associated with a *Bacillus coli* infection of the kidneys, and only after long continued treatment for the colon bacillus infection and for the acidosis did she get well.

Before we examined for acidosis, we have seen many patients in *status epilepticus* die some two or three days after the fits have ceased with rapid

respiration and pulse. This has also been pointed out by others and the cause of death has usually been attributed to "pulmonary congestion," but it may be that some of these patients die of an acidosis.

The patients whose cerebro-spinal fluid, blood or urine contained acetone, are shown in Table IV.

Discussion.

It is difficult to offer an explanation as to why a lumbar puncture should relieve a *status epilepticus*, whether arising from epilepsy, general paralysis or arteriosclerosis. In the majority of cases the fluid is under increased pressure and the usual amounts removed are from twenty cubic centimetres to sixty cubic centimetres.

The high pressure is more pronounced in the general paralytics and those with anteriosclerosis, than in the epileptics. In the two former or perhaps in all three the change in the fluid pressure and the change in the intracranial blood pressure that accompanies it, may bring about the result. The benefit obtained is so sudden, often immediate, that it is hard to attribute it to anything else than pressure. It seems unreasonable to attribute the result to removal of toxins, as this would be rather a blood than a cerebro-spinal fluid condition. The fluid is not highly toxic and its injection into animals does not invariably produce convulsions; in our hands animal injection never produces more than an indefinite and isolated attack.

In examining the urine of epileptics for chemical changes before and after fits, although no constant changes are noted, in the majority of cases we have found before attacks a diminished acidity, this often very low or an actual alkalinity, with a low ammonia content. Both the acidity and ammonia rise often to many times the normal after an attack or series and the more severe the series the greater the after acidity and the amount of ammonia excreted.

Bisgaard and Norvig[1] state that the ammonia content of the blood is up to three times normal within about three hours before an attack, whether a convulsion or psychic equivalent, and that there was low value after an attack. These authors found no important variations in the uric acid content of blood. They found a relatively low urea proportion of the total nitrogen and pH and ammonia nitrogen relations and they think the primary disturbance in epilepsy is an alteration in metabolism of the nature of an alkalosis. These findings as to the blood ammonia are of interest. Ammonia in very small doses will cause convulsions in guinea pigs. Some years ago we were endeavouring to give guinea pigs epilepsy by extracting a proteose from the blood of epileptics, using ammonium sulphate as a precipitant. The proteose was to be washed from the final precipitate on a small filter paper. The pigs had epileptiform attacks shortly after injection and some died. We then tried controls with solutions prepared in the same way, but *minus* the blood. These also had convulsions and died. The dose of ammonium sulphate they obtained was minute. The great increase of ammonia found in the blood by Bisgaard and Norvig may be the direct cause of the fit. After an attack or series things change round to the acid side.

It would appear as if the convulsions started with an alkalosis and ended in the more severe cases with acidosis. The acidosis is in no sense curative of the convulsive attacks, as we have seen in our cases quoted that the convulsions continue until the acidosis is cured and the acidosis adds an increased danger to an already dangerous illness.

Two of our epileptic patients, already referred to, have developed a severe and fatal acidosis during a quiescent period of their epilepsy. Is this a metabolic change dependent upon their epileptic condition and in some way an exaggeration of the chemical alteration that precedes or follows an attack?

The advocates of ketogenic treatment in epilepsy claim to prevent the attacks by a production of a ketosis and consider that diacetic acid is the active acid body in this respect.[2]

It seems as if the chief chemical changes before and after attack centre around the ammonia nitro-

Table IV.

Number.	Disease.	Fluid Examined.		
		Cerebro-spinal Fluid.[1]	Blood.	Urine.
32	General paralysis convulsions	+		
4	*status epilepticus* or series	+		
27	*status epilepticus* or series	+	+	
14	*status epilepticus* or series	+		+
26	*status epilepticus*	Not present	Not present; two days later +	
7	*status epilepticus*	+		
10	*status epilepticus*	+		
11	*status epilepticus*	+		
8	*status epilepticus*	First not present, later +	+	+
13	*status epilepticus*	+	+	+
12	*status epilepticus*	Not present		
53	Arteriosclerosis with convulsions	Not present		
52	Arteriosclerosis with convulsions	Not present		
	Primary dementia	Not present		
	Primary dementia	Not present		
54	Epileptic, no convulsions at time		+	+
	Epileptic, no convulsions at time			+

[1] + = acetone present.

gen metabolism. The organ most concerned with this is the liver.

Lalor[3] found the relation of the liver to the brain in epilepsy invariably reversed. He states that the normal liver and brain relation is 1,500 grammes liver and 1,200 grammes brain, with a wide margin on both sides. In epilepsy the relationship was altered, showing some atrophy of the liver. He quotes the comparative liver and brain weights, observed in twenty-five *post mortem* examinations of epileptics. In twenty of these the liver is lighter in weight than the brain. Lalor gives the brain-liver ratio as being 35 : 33 and the normal as being 12 : 15.

Pagnier[4] notes the "hæmoclastic crisis" occurring constantly in epileptics following injection of milk chocolate and Wilson notes the occurrence in eighteen of nineteen epileptics.

Acute destructive changes in the liver, brought about by such diseases as acute yellow atrophy or phosphorus poisoning, are followed by convulsions and the convulsions in Stokes-Adams disease may possibly be as much due to liver as to circulatory changes.

In a paper published in 1920[5] we described the ductless glandular changes found *post mortem* in sixteen cases of epilepsy; the liver in only one of these on microscopical examination was reported to be normal. Eleven were subject to fatty degeneration, one to cloudy swelling and three to fatty infiltration. Since then we have had an additional twenty cases in which the glands, including the liver, have been examined *post mortem*. In none of these has the liver been without pathological abnormality.

Table V. shows the naked eye and microscopical appearance of each of these livers.

These consistent *post mortem* changes are more than can be attributed to chance and it seems fair to presume some connexion between these changes and the disease.

Dr. V. M. Buschiano[6] states:

That protein disintegration might have some importance in relation to pathogenesis of epilepsy, was suspected by Rosenthal, based on his results on the antitryptic index of the blood serum of epileptics and by Tuschtschenko who thought that the epileptic seizure was due to an accumulation of abnormal katabolic protein products through insufficiency of the thyroid function.

Cuneo noticed the presence of materials of protein disintegration in the blood, (albumose) and thought possible a toxic origin of epilepsy from this substance.

Buschiano gives his opinion:

As the result of the comparison of the biological characters of the epileptic seizure and the anaphylactic crisis is such as one can confidently affirm that the epileptic . . . seizure presents the characters of an anaphylactic crisis and we can affirm that the epileptic seizure like the anaphylactic crisis is an intoxication with substances belonging to the group of peptones or with other substances derived from the breaking down of proteins.

Table V.

Case.	Age.	Macroscopical Appearance of Liver.	Microscopical Appearance of Liver as Reported by Dr. O. Latham.
A.			A little fibrosis of portal system, some narrowing of lumen, some brown pigment in cells of hepatic vein zone.
B.	27	3 lbs., pale and fatty; brain 2 lbs.	The nuclei are preserved, a little brown pigment in hepatic vein area and quite a considerable fatty infiltration in the portal zone, capsule thickened slightly, but not very fibrous.
C.	64		Slight fibrosis of portal canal, some fatty degeneration.
D.	20	Fatty	Fatty globules in portal zone. Hæmatoid pigments central zone, with œdema and signs of very chronic congestion.
E.	31	Small, smooth and many patches of fatty degeneration	Fatty globules in portal zone. Hæmatoid pigments central zone, with œdema and signs of very chronic congestion.
F.	45	Large and congested	Intense fatty degeneration of hepatic vein area with intense engorgement. Some brown pigmentation vacuolation of the nuclei.
G.	30	Extremely congested and fatty	A slight interstitial increase. Liver cells show cloudy swelling (fatty degeneration), there is œdema separating the column of cells.
H.	23	Liver 45 ozs.; brain 53 ozs. ..	Slight increase of round cells in portal area, some œdema in *zona hepatica* and some fatty degeneration.
I.	30	Congested and fatty	Some pigment and œdema in the hepatic vein system.
J.	44	Large and firm, fatty bladder sclerized	Œdema and brown pigment venous zone, some fatty degeneration of the cells. Capsule thickened and cells of bile duct trifle proliferated. A little fibrosis of portal canals.
K.	34		Full of granules, some cloudy swelling and fatty infiltration, considerable increase of old fibrous tissue round portal tracts, especially of the large bile ducts.
L.	35	Large, firm and congested 62½ ozs., brain 52¾ ozs.	
M.	35		Some œdema and fatty degeneration. Nuclei well stained, cells compressed.
N.	55	Liver fatty, old hydatid cyst in right lobe. Weight with growth 68 ozs.; weight without growth 49 ozs.; brain 47 ozs.	
O.	47	Fatty.	
P.	63	Small and pale.	
Q.	60	Fatty	Cells of liver pale, fatty infiltration, interstitial and intercellular tissue œdematous, small amount of brown induration near hepatic vein zone.
R.	57	Congested weight 2 lbs. 15¼ ozs.	Intercolumnar interstitial tissue is œdematous and some brown pigment in hepatic vein zone in the cells. A small amount of fibrosis rather hyaline.
S.	65		Some old pigment in hepatic vein zone, a little very chronic congestion, some œdema hepatic vein zone.
T.	32		Fatty infiltration hepatic vein zone, with some brown pigment granules. Passive venous congestion, slight fibrosis portal canals and interstitial tissue carrying bile ducts.

Buschiano denies that the protein or peptones which he thinks causes the anaphylactic reaction, are found or formed in the intestinal tract, but says that in epileptics there is an abnormal protein derived from the thyreoid. These pass into the circulation at intervals and if this occurs after sensitization, peptones or like toxic substances are set free.

In our own series of twenty-three thyreoids from epileptics examined by Dr. Evan Jones, no constant change was found, five being described as normal, the others showing well marked changes of hyper- or hypothyreoidism. While feeling much admiration for Buschiano's work, one cannot help feeling that if epilepsy is a result of anaphylaxis, it is more probable that the toxin is derived from the alimentary canal and is the result of hypofunction of the liver, this organ failing to convert some of the protein derivatives into urea. In favour of this idea is the result of our examination of livers just mentioned and also the effect of any digestive upset or indiscretion in diet. Want of moderation in taking alcohol, very rich food or constipation in an epileptic will almost invariably bring on an attack. Often a soap and water enema, given in a case of *status epilepticus* or serial epilepsy, will cause for the time an absolute cessation of the attacks Moreover, whatever is given in these conditions, there will not be any beneficial result until after a free purgation, showing a very intimate relationship between the digestive system and the epilepsy. Further, some of the derivatives of protein digestion, such as guanidine and histamine, are known to be convulsive, as is also ammonia and some of its compounds. Sodium biborate is perhaps after the bromides the most useful drug in the treatment of epilepsy. Hale White[7] states that it increases the amount of urea eliminated and the quantity of urine passed and that it also diminishes the acidity of this fluid. If so, it would seem that its usefulness is due to its hydrolysing into urea one of those ammonia compounds or amino acid that may be the toxic agent in epilepsy.

Summary.

1. Lumbar puncture is an effective treatment in *status epilepticus*, our series showing a recovery rate of 95% in so-called idiopathic epilepsy.

2. It is also of value in convulsions due to arteriosclerosis and general paralysis of the insane.

3. Acidosis is common in *status epilepticus* and the same metabolic disorders may cause death without convulsions in epileptics.

4. It is suggested that the basis of epilepsy may be an interference with the detoxicating functions of the liver, allowing the toxins of normal or abnormal alimentary functions to reach the systemic circulation.

References.

(1) A. Bisgaard and F. Norvig: "Further Investigations in Regulations of Neutrality in Epilepsy," *Zeitschrift für die Gesellschaft der Neurologie und Psychiatrie*, Abstract in *The Journal of Mental Science*, January, 1924, page 143.

(2) M. G. Paterman: "Ketogenic Diet in Epilepsy," *The Lancet*, August 23, 1924, page 402.

(3) Peter Lalor: "Toxæmia in Epilepsy," The Medical Journal of Australia, March 20, 1920, page 251.

(4) Isabella M. Robertson: "Vasomotor Actions in Mental Diseases," *The Journal of Mental Science*, October, 1925.

(5) G. P. U. Prior and S. Evan Jones: "Some Mental Cases with Endocrine Considerations," *The Journal of Mental Science*, January, 1920.

(6) V. M. Buschiano: "Epileptic Seizures and Anaphylactic Crises."

(7) W. Hale White: "Materia Medica," Volume II.

HYPERTROPHIC PYLORIC STENOSIS AND PYLORIC SPASM.[1]

By A. H. P. E. Verbrugghen, M.B., Ch.M. (Sydney),
Guyra, New South Wales.

Before I commence I wish to make it perfectly clear that the paper which will follow, is not by any means original. I had difficulty with a case which turned out to be a severe example of pure pyloric spasm. I was at sea and the child was obviously going to die, so I sat down to see what I could make of it and this is the result. As far as I can see the only thing which might commend this paper, is that a whole lot of scattered facts have been drawn together to bear upon one point. As far as I know these facts have not been connected before. The subject is now very much clearer to me and I hope that I may perhaps place you in the same position.

Ætiology: Various Theories.

The first theory is that the condition is a primary congenital hyperplasia and that the abnormalities in function are the result and not the cause of the muscle enlargement (Hirschprung and Cauteley) that is, that the condition is a congenital anomaly like club foot.

The second theory is that the condition is not a congenital malformation, but is consecutive to spasm of the pylorus and that the great severity and the long continuance of the spasm lead to hypertrophy as in any other muscle. There are as far as I can find five views as to the causation of this spasm which goes on to hypertrophy.

(*a*) Some say that an incoordination exists long before birth and that it increases when feeding with milk is begun. These people believe that the hypertrophy is not very pronounced at birth.

(*b*) Some say that the spasm is brought about by an excess of acid in the stomach contents, because they believe that excess of acid in the stomach leads to closure of the pylorus. This might be differently expressed in the following manner: That the source of the hyperacidity and the source of the stimulation of the pylorus is a common one. I myself believe that the pylorus is caused to contract through over activity of vagus nerve which would also cause hyperacidity. I hope to make this clear later on.

(*c*) Others say that there is something wrong with the chemical mechanism of the duodenum which Pawlow has shown to be responsible for a normal opening and shutting of the pylorus.

[1] Read at a meeting of the Northern Districts Medical Association on January 23, 1926.

(*d*) There is another suggestion that the spasm is due to an excess of adrenalin in the blood which was first present before birth, that is, "prenatal hyperadrenalism."

(*e*) Barr says that the condition is not congenital being "due to an excessive amount of calcium salts in the stomach walls and especially in that portion of the walls, the pylorus, which contains relatively the most muscular fibres." Involuntary muscular fibres are powerfully influenced by soluble calcium salts and therefore, when the salt is present in excessive amounts, spasm of the pylorus muscle is induced. Doubtless growing infants require a large amount of lime, but apparently even infants may have too much of a good thing.

In connexion with these widely divergent views on the ætiology of the complaint one must consider two very important clinical facts: (i.) The symptoms do not arise immediately after birth but generally a fortnight or more later. (ii.) That the condition is capable of being absolutely cured by purely medical treatment leaving the stomach in a perfectly normal condition afterwards.

Taking consideration of these two facts it would appear that the condition, at first at any rate, is made up of two factors, a functional and an organic, and that the various proportions of these two factors determine the severity of the condition. If the stomach is capable of being absolutely returned to normal by medicinal treatment only, then the condition is not organic to any large extent, but must be to a certain extent functional.

With all these theories of the ætiology the weak point lies in why it takes two or three weeks from birth for the symptoms to develope. This weakness is particularly noticeable in the congenital hyperplasia theory. Of course, any of these suggestions may be true but there is as yet insufficient evidence to warrant their being wholly accepted.

From a common sense point of view and also from the point of view of treatment there is only one thing to believe, that is, that, explain it as you may, an incoordination exists with resultant spasm of the pylorus which goes on to hypertrophy and stenosis. These views will be reconsidered when the question of treatment is under discussion.

There are cases of pure pyloric spasm which occur as clinical entities and which never go on to hypertrophy.

Morbid Anatomy.

The essential structural charge is confined to the upper portion of the alimentary tract.

It consists of: (i.) A pure hypertrophy of the circular muscle of the pylorus and hypertrophy of the stomach wall; (ii.) a secondary dilatation of the stomach in proportion to the amount of obstruction; (iii.) a chronic gastric catarrh secondary to pyloric hypertrophy.

Microscopical examination reveals hyperplasia of the circular muscle only. The tumour forms a dense elastic swelling bulging in the centre.

The peritoneal coat is distended to its utmost limit and is so tightly stretched by the muscle as to be circular in transverse section. This inability to stretch, caused by non-elastic peritoneum, causes the mucous and submucous coats to be compressed and apparently thickened. This also increases the obstruction. This second factor does not come into play in the earlier days of life, but only after the second or third week or later, when the muscle is considerably thickened. Consequently only then do the symptoms of severe obstruction appear and the difference in the rate of the muscular hypertrophy in different children is the explanation of the varying dates of onset of urgent symptoms.

Some authorities believe that there is an adenomatous enlargement of the mucous membrane concomitant with the muscle hypertrophy.

The essential disturbance in function is an ill-timed forcible contraction of the pylorus. In the cases of pure pyloric spasm there is disturbance of function only and there is little or no muscle hypertrophy. So pyloric spasm can be explained as a disturbance of function only, not going on to hypertrophy and pyloric stenosis is a spasm in the first place which goes on to hypertrophy causing stenosis.

General Considerations: Nervous Mechanism of the Stomach.

In my opinion it is impossible to understand properly either pyloric spasm or stenosis without some reference to the nervous mechanism of the stomach.

A local mechanism is maintained through the presence of Auerbach's plexus of nerves situated between the longitudinal and circular muscle layers of the stomach; intimate connexion with the great nerve centres is obtained through the fibres of the vagus and sympathetic nerves. These two nerve sets must be distinguished. The sympathetic proper includes fibres taking exit from the thoracic and lumbar regions of the cord which supply blood vessels, glands and smooth muscle fibres of the body, including those of intestinal tract, liver and pancreas. The vagus system supplies the glands and muscles of the gastro-intestinal tract, liver and pancreas. It has been suggested that these two systems are capable of being excited, leading to definite symptom complexes. Stimulation of the vagus system has been termed vagotonia and stimulation of the sympathetic system, sympatheticotonia. Excitation of the vagus leads to increased gastric secretions (hyperacidity), increased peristalsis, cardiospasm, pylorospasm and spastic constipation *et cetera*. Excitation of the sympathetic would lead to atony of the intestinal tract with constipation, ptosis, *achylia gastrica* and nervous anacidity.

It is further known that certain drugs have a definite effect of stimulation on the vagus nerve while others have an inhibitive effect. The same applies to the sympathetic system. According to Cannon the presence of free hydrochloric acid in the stomach causes the relaxation of the pyloric

sphincter which allows the acid chyme to escape into the duodenum. The presence of acid in the duodenum produces a reflex stimulation of the pylorus and this makes it close and remain so until the contents of the duodenum have again become alkaline. Without doubt the sphincters of the stomach are also controlled by the nervous system through the vagus and sympathetic nerves.

Another fact which bears on pyloric stenosis, is that peristaltic waves are not present in the cardiac end of the stomach, whereas, during digestion, there are constant peristaltic movements running over the surface of the pylorus. Meltzer pointed out the law of contrary innervation of the intestines which exists even in voluntary muscle. According to this law stimulation of the intestine causes a contraction above and an inhibition below the area of stimulation. Further it has been shown by experiments that stimulation of the vagus causes contraction of the pylorus.

These considerations have led me to believe the following:

(i.) That in pyloric spasm there is a definite over stimulation of the gastric fibres of the vagus causing a functional disturbance.

(ii.) The pyloric stenosis is secondary to spasm of the pylorus and is really a later manifestation of the same complaint.

(iii.) That there are cases of pure pylorospasm recorded which never go on to stenosis, as the majority do and that these cases are due to an intermittent, as opposed to a continuous spasm, for example, spasmodic diarrhœa.

Expressing this in another way, I should explain pure pyloric spasm as a true incoordination causing the breaking down of the law of contrary innervation, that is the pylorus does not relax when the stomach contracts. Those conditions, however, which we afterwards know as pyloric stenosis, are due to a continuous over stimulation of the vagus causing a continuous spasm which naturally goes on to hypertrophy.

So it would appear from the above that pure pyloric spasm and pyloric stenosis have a somewhat similar origin. It must be clearly understood that this is the only way in which I could find an adequate explanation of the two conditions for my own edification. This is the attitude which I am adopting towards this illness in this paper, that is, that pyloric spasm and pyloric stenosis are practically different stages of the same thing.

Symptoms and Physical Signs of Pyloric Stenosis.

The condition is more common in boys than in girls, according to Still in the proportion of 1 to 6·78 and more common in first-born children. Half of Still's patients were first-born children. The children came of normal or more often distinctly nervous parents. Instances are on record of two children in one family being affected. In my own experience I have known this to occur.

The first child, a girl, died after operation for stenosis and the second child, a boy, has been under my care suffering from a particularly severe form of pure pyloric spasm. The father was a stutterer.

The main symptoms are three in number, vomiting, wasting and constipation. The vomiting has several peculiar characteristics. It is extremely violent and has been called projectile. By this is meant that the child will suddenly and without warning throw up its food with such force that the food is shot out from the body sometimes two or three feet. Some of the vomit goes through the nose. The vomit is also characterized by the fact that usually the child throws up more food than was taken at the last feed, that is, the child gets one or two feeds down and has apparently digested them when suddenly it vomits the lot. This shows that there is a retention of food in the stomach. There is frequently mucous in the vomited material and a change of food frequently stops the vomiting for twenty-four hours, but it recommences.

The motions are small and usually dark in colour. The urine is highly coloured and scanty. In other words the child is very constipated and may only wet two or three napkins in twenty-four hours.

The child is exceedingly hungry. The expression is pained, but alert. These symptoms are also typical of pure pyloric spasm.

The physical signs are: Emaciation, visible peristalsis, palpable pyloric tumour, gastric dilatation.

The emaciation is obvious. The child has an abdomen which is described as pear shaped, that is, full in the hypochondriac and epigastric regions and very small in the iliac and hypogastric regions, the skin which hangs on the child in wrinkles and folds, is usually dry. The fontanelle is sunken, but in spite of all this the child looks bright. The emaciation is present in the pure spasm, only it is not so pronounced. In severe cases of pure spasm the emaciation would be very pronounced.

Visible peristalsis will be noted principally when the stomach is full, but may be seen at any time. The time to look for it is when the child is being fed. The waves will be seen arising under the left costal margin and travelling after one another to the right hypochondrium. Some authors describe this as pathognomonic of the disease. In typical cases it is quite definite and can be seen from the other side of the room. In pure spasm this peristalsis is much less pronounced and may be absent.

The presence of palpable pyloric tumour is really the only pathognomonic sign of pyloric stenosis. With warm hands and with the child comfortable and at rest the tumour may be felt above and to the right of the umbilicus. It may possibly be felt contracting and relaxing, if it is picked up between the fingers and thumb, though this may not be possible. It is about the size of a walnut or a pigeon's egg. Occasionally this tumour cannot be felt owing to the pylorus being tucked up under the liver. In true pyloric spasm there is no tumour.

Gastric dilatation may be demonstrated by washing out the stomach which may contain one hundred cubic centimetres of fluid or more. This condition, I should think, would be present only late in the stenosis when the obstruction is fairly complete. In

the early stages of pure spasm the same condition holds, but in the late stages of pure spasm the stomach may be definitely dilated.

In a given case of pyloric stenosis the severity of the symptoms would probably vary in proportion to the amount of functional as opposed to organic abnormality. The more pronounced the organic stenosis, the more prominent would be the symptoms. Severe forms of purely functional spasm give rise to severe symptoms, as in organic stenosis.

It must be understood that in comparison to pyloric stenosis pure pyloric spasm is very rare. To sum up, the symptoms of pure pyloric spasm are the same exactly as in stenosis. As regards physical signs there is no tumour and little, if any, visible peristalsis is to be seen.

Diagnosis of Pyloric Stenosis.

There are only two signs which can be regarded as pathognomonic, pyloric tumour and visible peristalsis. Of these the tumour is far the most important. Can the peristalsis be taken for any other movement? The visible peristalsis may be mistaken in the first place for movements of the abdominal muscles when the child is squirming about. There are very rare instances in which owing to derangement of the parts peristaltic movement of the transverse colon has been mistaken for that of the stomach. The peristaltic waves of the colon are from right to left in the normal position. Visible peristalsis does occur in the rare conditions of stenosis of the duodenum and obliteration of the duodenum, but here the symptoms will put one right. The symptoms appear in the first day or two of life and the vomit would contain bile. Chronic gastric catarrh often causes difficulty in the diagnosis before the tumour can be felt and before the peristalsis is present. Chronic gastric catarrh is particularly difficult to distinguish from pure pyloric spasm, especially if the symptoms have just made their appearance. In the diagnosis between pyloric stenosis and pure pyloric spasm it may be remembered that in the former the tumour and the peristalsis are present, whereas in the latter there is no tumour and little peristalsis. But even here difficulty may be experienced as a pyloric tumour might be tucked up under the liver and inaccessible to the palpating fingers. Fortunately this tucked up condition is not frequent. All the other symptoms and signs are the same in stenosis and pure pyloric spasm. The vomiting and constipation of pyloric obstruction have been mistaken for cerebral disease.

Prognosis.

The prognosis in pyloric stenosis has been greatly improved by early operation using Rammstedt's method. This will be discussed more fully later. The prognosis in pure pyloric spasm is supposed to be very favourable.

Treatment of Pyloric Stenosis.

The medical treatment of pyloric stenosis is the same exactly as the treatment of pyloric spasm. No further attention will be drawn to this point.

Diet.

All authorities are agreed that under no circumstances should the child be weaned, the breast is naturally the best food for the sickly child and breast milk does not give rise to the large curds of cow's milk and other artificial foods, it is consequently the more able to pass through the constricted pylorus.

If the child has been unfortunate enough to have been weaned, there will be great difficulty in finding a suitable food. All sorts and conditions of foods have been recommended, Robert Hutchison suggests fully peptonized milk diluted with an equal quantity of water. The people who believe in the calcium salt theory, give citrated milk (one grain to the ounce of sodium citrate). Should these fail one is advised to try feeding on a whey basis or whey and cream, whey and egg white, desiccated milk, malted milk and so on. No doubt there are practically never ending combinations that could be used and have been used.

In my opinion the food, if it be artificial, should be such as will easily pass through the narrow pylorus, that is, the food must form only very small curds in the stomach. For this reason whey and Mellin's food form a very suitable combination. Such mixtures as peptonized and citrated cow's milk form fairly large curds and do not agree for that reason. It is quite conceivable that a large curd lodging in the narrow pylorus would not only temporarily increase the obstruction, but in addition would cause spasm by irritating the pylorus. Therefore it is suggested that whey and Mellin's food be used. I have seen this food returned after it had been two or three hours in the stomach and the consistency of the food was practically the same as when it was taken. It is prepared by using whey instead of the milk and water in the printed directions.

Then there is the question of the interval between feeds and you are recommended to be guided by the capacity of the stomach. I should say that it would be fairly hard to estimate the capacity of the stomach. As far as I am concerned I have been able only to hazard a guess in my cases and that more by external percussion than by the use of the stomach tube. Apart altogether from the size of the stomach some men recommend feeds every four hours, others every three hours and they suggest that the feeds should be small. Others suggest that the feeds should be small and frequent.

The interval of feeding and the quantity of the feed will differ in all cases. I believe that small frequent feeds are the best, thirty to forty-five cubic centimetres (an ounce to an ounce and a half) every hour or hour and a half. Each patient will have to be experimented with until a suitable arrangement is reached. From the point of view which I have of these patients I do not think that this feeding question is the most important point for reasons which will be explained.

Children with this condition are suffering from an obstruction and the way in which they are fed,

will not remove that obstruction. Certainly judicious feeding may cause a certain amount of food to insinuate itself through the unwary pylorus, still I feel that the aim of treatment should be at the obstruction and with that even partly relieved, the question of feeding loses a great deal of its importance.

Stomach Washing.

Next in order of merit in the textbooks comes stomach washing as a remedy in pyloric stenosis. What is used for the actual washing is immaterial, but usually a weak solution of sodium bicarbonate is advised. All authorities agree that stomach washing is a very necessary part of the treatment and they nearly all recommend it to be done twice a day. I presume the rationale of stomach washing is to remove the curds collected in the stomach owing to the obstruction at the distal end and also to remove any mucus that may be present in the stomach.

The mucus would not be present in the early stages of the disease, but only when the stale stomach contents continually collecting would have had time to set up a chronic catarrh. This treatment would also be more or less unnecessary when something had been done in restoring the patency of the pylorus. Stomach washing is treatment aimed at the effect of the disease and not at its cause. It is used to remove stale food, so that fresh food may not be contaminated, so that it may have a chance of passing through the pylorus. Stomach washing then has no effect on the actual obstruction; on the contrary it appears to me that it would increase the spasm. It seems only reasonable that passing a stomach tube into the stomach would by exciting it tend to increase its activity. Even passing a tube down the throat has a similar effect. While the obstruction persists, any one will freely admit that stomach washing is necessary; but as far as the actual obstruction is concerned it does not help, in fact it makes it worse temporarily. It must be assumed therefore that patients with pyloric stenosis who recover under the treatment of stomach washing and diet, do so by the fact that the stenosis gradually cures itself and that in the meantime the treatment enables sufficient food to pass the pylorus to keep the child alive. It may be claimed that this is all that is required, but it is not; all treatment should be directed at the cause of a complaint and not at the effect.

The Use of Saline Solution.

The administration of saline solution is advised by some authors and not by others. It is mainly used now as an adjunct to surgical treatment before and after operation. It can be given *per rectum,* subcutaneously or intraperitoneally. In this disease the usual method is by subcutaneous injection into the skin of the abdomen. It can be given in the back, but this is objectionable for the child cannot be put down in a comfortable position. The use of these injections should depend upon the state of the child and is indicated when there is much dehydration of tissues and when very little urine is being passed owing to persistent loss of fluid.

Drugs.

None of the authorities on diseases of children whose books I have read, place much reliance in drugs. They have no faith in belladonna and very little in opium. Of the two they prefer opium. Citrates are recommended by those who cleave to the calcium salt theory of the causation of pyloric stenosis. This lack of faith in drugs, when admitted by such eminent authorities, makes me hesitate in saying what I have to say, namely, that large doses of atropine have acted like a charm in my cases; large doses let it be noted, for I believe that small doses only make matters worse. This treatment has been attempted only in two cases, one of pure pyloric spasm and one of early stenosis. On going into the anatomy and pharmacology of the subject large doses of atropine would be expected in the ordinary way to relieve spasm of the pylorus. The bearing which this treatment has on pyloric stenosis, depends on an opinion which I have already expressed, namely, that in any given case of stenosis there is a functional and an organic factor.

If the organic obstruction is not too far advanced, a reduction or cessation of the spasm ought gradually to effect a cure. For, the spasm being relieved, the hypertrophy far from progressing should retrogress. Any treatment which aims at relieving either the functional or the organic factors or both factors is to be highly commended and appears to me to be far more rational than treatment directed at the effect of the complaint.

From the pharmacologists we learn that large doses of atropine paralyse the vagus nerve and so abolish peristalsis and spasm of the pylorus. From the same source (Hale White) we also learn that small doses of atropine increase peristalsis, supposedly by stimulating the plexuses of Auerbach. This characteristic of the drug is of the greatest importance in its administration especially in pyloric spasm and stenosis, for small doses according to this would tend only to aggravate the condition.

In a patient with pure pyloric spasm under my care small doses of atropine (0·32 milligramme or one two hundredth of a grain divided into seven doses per day) were given and the effect was absolutely nil. Later large doses were given and they acted like a charm.

I would suggest then that the drug be given in large doses and if necessary up to the point of dilatation of the pupils and flushing of the face. At this point it should be temporarily ceased. I should describe a large dose as two drops of a one in a thousand solution of atropine sulphate in water. This can be given five or six times a day, in fact half an hour before every feed. This interval before food is selected so as to give the atropine time to act. As children stand atropine well, it can be pushed to the point of toxic action.

One of my patients at the present moment is receiving double this dose before every feed. The tolerance to the drug is complete. The child does not vomit except perhaps once in forty-eight hours. A few days ago a test was made and the atropine was discontinued for twenty-four hours. During that day the child vomited five times in the most characteristic projectile fashion. The next day he was given three drops of the solution before each feed and that day he vomited twice. The next day he did not vomit at all. This was a costly experiment. Those two days cost a nine and a half pound child half a pound.

Atropine is given to adults for conditions due to over activity of the vagus. It is given to prevent the effect of excitation of the vagus on the heart when administering chloroform. Atropine is used with success in certain forms of heart block owing to its paralyzing the vagus nerve. Rodgers has given atropine in large doses to people suffering from over activity of the gastric fibres of the vagus, causing hyperacidity, cardiospasm and pylorospasm. The administration was said to have a very good effect. I have given it to a patient suffering from entero-spasm. However, this case of entrospasm was very interesting and if I may be permitted, I may read a description of it at some other time.

Under these circumstances one would expect atropine to be useful in the cases of pyloric stenosis in which there is still a large element of spasm. Johanennsen has reported seven cases of pyloric stenosis in which the administration of large doses of atropine effected cures. In four of the cases the suspension of the drug was followed by a return of the vomiting and this was abolished by the renewal of the treatment. He says the full effect of the drug was deferred until the tenth to fourteenth day. In three tolerance to the drug was complete, in three transient redness of the face and dilatation of the pupils were observed and in one the toxic action of the drug caused it to be temporarily abandoned. For pure pyloric spasm this would appear to be the treatment *par excellence* and in my case of pyloric spasm which I described above, it certainly acted like a charm.

Of course it is obvious that if the condition has progressed until the obstruction is mainly organic and almost complete, then the condition has passed out of the realm of the functional and has become practically a mechanical affair and only mechanical treatment will relieve it, that is, operation.

Opium is sometimes useful, but it is a dangerous drug to give to weakly infants. The dose recommended to be given in pyloric stenosis and pyloric spasm is from 0·001 to 0·01 milligramme (one-sixtieth to one-fifth of a minim) of tincture of opium half an hour before a feed.

Surgical Treatment.

The operations for hypertrophic pyloric stenosis are four in number: (i.) Rammstedt's operation; (ii.) gastro-jejunostomy; (iii.) pylorodiosis (Loreta's operation); (iv.) pyloroplasty.

It is unnecessary to describe these in detail as they are known to everyone. The essential feature of Rammstedt's operation is that the serous and muscular coats of the hypertrophied pylorus are divided, but are not sutured. The mucous membrane is not opened.

Gastro-jejunostomy explains itself.

Loreta's operation is performed by opening the stomach proximal to the pylorus and passing sounds of gradually increasing size through the pylorus until the muscles and serous coat are stretched and torn sufficiently to prevent the narrowing of the pylorus from occurring again.

Pyloroplasty is very similar to Rammstedt's operation, but all the coats of the stomach are divided longitudinally and sutured in a transverse direction.

Whatever operation is undertaken it has been stated that it should not take longer than fifteen minutes, but I take it that only Rammstedt's operation can be meant, as none of the others could be done in that time except perhaps by one exceptionally endowed with much experience and most agile fingers.

The most suitable incision is through the right rectus above the umbilicus; the midline is weak in children and there is a definite tendency to incisional hernia.

Comparative Value of the Various Surgical Operations.

Pyloroplasty is practically Rammstedt's operation *plus* opening the mucous coat and suturing all layers of the bowel in a transverse direction. The suturing makes the operation long with no additional advantage and opening the mucosa makes a risk of soiling the peritoneum.

Loreta's operation also involves the risk of soiling the peritoneum through opening the stomach mucosa. Both the time to be taken at the operation and the degree of rupture of the muscular and peritoneal coats is uncertain and there is a risk of injuring the mucosa. Scar-tissue is reported to have formed at the site of operation causing a traumatic pyloric stenosis.

Gastro-jejunostomy has a high mortality. The operation is difficult in the adult and very difficult in the infant, the tissue being fragile and the sutures having a tendency to tear out. Peritonitis, an inadequate stoma and the time factor are the principal causes of the fatal results.

Rammstedt's operation is the most suitable operation. It is not very difficult, it can be done quickly and if properly done there is no risk of fouling the peritoneum. The results are excellent.

Postoperative Treatment.

Postoperative treatment is very important. The infant must be kept warm and subcutaneous saline solution must be administered should the child's condition be unsatisfactory. With regard to the feeding after operation various writers have different views. One section adopt the following plan: If the child be breast fed, four cubic centimetres (a drachm) of breast milk are drawn off and given to the child after operation. This is increased until, at the end of the third day the child is put on the

breast every three hours for fifteen to twenty minutes. With the bottle fed children they use a reliable skimmed dried milk in much the same way until at the end of the third day the child is taking from seventy-five to ninety cubic centimetres (two and a half to three ounces) every three hours.

Another section of medical men use the following scheme: In breast fed babies they give eight cubic centimetres (two drachms) of breast milk with eight cubic centimetres of boiled water every two hours for twenty-four hours. Then the next day they give fifteen cubic centimetres (half an ounce) of breast milk and the same quantity of boiled water alternatively every two hours. In bottle fed babies they use peptonized milk in the same way as they use the breast.

Mortality from Operations.

It is a very hard matter to discuss operation mortality, as there have been four different operations in use. I believe that they are now nearly all abandoned in favour of Rammstedt's operation. On examination of the mortality lists given below it will be agreed that this is as it should be, Rammstedt's method yielding a considerably lower mortality than any of the other methods.

By far the most instructive mortality list I could procure was that of L. E. Holt and fortunately they are the cases of one surgeon, W. A. Downes, of New York. He divides these cases into four periods. These are shown in the accompanying table:

Table Showing Results Obtained by W. A. Downes of New York.

Treatment.	Periods of Time.			
	1901-11.	1912-14.	1915-16.	After 1916?
Without Operation	24 Cases Mortality 58%	7 Cases Mortality 57%	2 Cases Mortality 50%	—
Gastro-enterostomy	17 Cases Mortality 58%	24 Cases Mortality 50%	—	—
Rammstedt's Operation	—	6 Cases Mortality 33%	61 Cases Mortality 23%	175 Cases Mortality 17%

Since 1916 he has operated on one hundred and seventy-five patients with a mortality of 17% and he states that if the patients are operated on under four weeks after the onset of symptoms that the mortality is 8%.

John Thomson quotes one hundred cases as follows:

Gastro-enterostomy mortality	64%
Loreta's operation mortality	61%
Rammstedt's operation mortality	20%

Tyrrell Gray quotes for Rammstedt's operation only:

All cases mortality	41%
Uncomplicated mortality	23%
Favourable (early) mortality	9%

T. T. Higgins quotes fifty-five cases of Rammstedt's operation:

All cases mortality	29%
Sixteen cases mortality	20%
Sixteen cases (in last 2½ years) mortality	13%

These lists ought to be quite sufficient to show that Rammstedt's operation is the only method to be considered as far as surgery is concerned.

Mortality from Medical Treatment.

It seems very hard to determine the mortality from medical treatment and Still is the only man to whose statistics I have access, the others do not quote. It is possible that I have made a mistake and included in the mortality all his cases in which treatment was by both medical and surgical means. This improves considerably the mortality of those treated by medical means. I make the best mortality from medical treatment on his figures 35%, but Holt is apparently unable to do better than 50%.

So of all the methods of treatment both medical and surgical the best chances of recovery are afforded by Rammstedt's operation. If this operation be done early, the mortality is somewhere between 8% and 13%, whereas the mortality from medical means is at least 35%. The disease is consequently no longer a medical disease but a surgical one and it is unjustifiable to persist with medical treatment. It is just as unjustifiable as to persist in applying hot fomentations to an acutely inflamed appendix, although frequently acute attacks do subside under this treatment.

Conclusions.

(1) Hypertrophic pyloric stenosis is not congenital.

(2) In hypertrophic pyloric stenosis there is an initial spasm which is continuous and which goes on to hypertrophy.

(3) The spasm is caused by over stimulation of the vagus nerve.

(4) In pure pyloric spasm the spasm is intermittent and is more in the nature of a true incoordination, as in spasmodic dysmenorrhœa.

(5) On no account should any child suffering from vomiting be weaned until hypertrophic pyloric stenosis has been completely excluded from the differential diagnosis.

(6) Treatment, whether medical or surgical, must be directed to the cause, that is, an attempt should be made to paralyse the vagus nerve in the hope of relieving the spasm. This may succeed should the hypertrophy not be too far advanced.

(7) Large doses of atropine paralyse the vagus and relax the pylorus.

(8) In pure pyloric spasm atropine in large doses is the most suitable treatment.

(9) In pyloric stenosis medical treatment should not be persisted in, if improvement is not seen in

six or seven days, as delay and medical treatment are far more dangerous than operation.

(10) The most suitable operation is Rammstedt's operation.

(11) The only sure diagnostic sign is pyloric tumour and operation should rarely be undertaken in its absence.

Reports of Cases.

LAWN TENNIS INJURIES.

By J. M. Baxter, M.D. (Melbourne),
Surgeon to Ear, Nose and Throat Department, Saint Vincent's Hospital, Melbourne.

Although tennis is a very popular game, serious injuries to the eyes or ears are not very common. Yet during the first six months of this year, 1926, I have met with four cases, three of which had had unfortunate endings.

Case I.—Miss B., aged thirty-one years, whilst playing tennis was struck on the right eye by a swift ball. After a couple of minutes' rest she played on, but on returning home she found she was practically blind. There was no external evidence of injury. The region of the macula was covered by an extensive hæmorrhage. There has been no improvement in vision.

Case II.—Miss X., aged thirteen years, was struck by a tennis ball on the left eye. The eye was very painful on examination. The pupil was fixed and widely dilated. The retina on the nasal half was extensively torn and accompanied by hæmorrhage. Practically no vision has been retained and the pupil remains dilated.

Case III.—Mr. C., aged thirty-five years, a farmer, whilst trying to avoid a swift return was struck on the right ear. Apart from a transient pain he was unaware of any injury till his return home when he found his hearing was very dull in the injured ear. The drum was ruptured in the posterior quadrant, the rupture extending vertically almost the full diameter. The ear suppurated profusely and three months later there were symptoms of mastoid development.

Case IV.—Mr. R., aged twenty-three years, was struck on the right ear by a tennis ball. There was much pain and he was very dazed. On attempting to rise from the ground he had intense vertigo and vomiting. He was removed to private hospital. There was a small perforation in the posterior inferior quadrant of the drum and a fair degree of hæmorrhage. During the first week any movement of the head produced vertigo and vomiting. In about ten days the perforation had healed and vomiting had ceased, but the vertigo persisted for six or seven weeks. The hearing which at first was reduced by one-half, is now almost normal.

A CASE OF CARDIAC MASSAGE WITH RECOVERY.

By Leo Doyle, M.S. (Melbourne),
Surgeon to Out-Patients, Saint Vincent's Hospital, Melbourne.

Mrs. R., aged thirty-six years, was referred to me on March 3, 1925, suffering from trigeminal neuralgia affecting mainly the maxillary and mandibular divisions of the nerve. About seven days later these two branches were injected with alcohol. This gave a good result, namely freedom from pain for six months. Then after a few months of gradually increasing pain she again saw me about obtaining relief. She now elected to have a radical operation with the expectation of complete and permanent relief rather than the temporary relief afforded by injections.

I wished to perform the operation as far as possible under local anæsthesia, but as no amount of persuasion would bring her to see the advantages of this, it was decided to use general anæsthesia.

On February 24, 1926, I set out therefore to divide the posterior root of the trigeminal nerve and ether was administered by the intrapharyngeal method through a nasal tube. The patient was placed in a semisitting position and the operation commenced. It proceeded satisfactorily but for the hæmorrhage which was troublesome rather than dangerous. Some trouble was met in the floor of the fossa, as there was a bony ridge blocking access to the middle meningeal artery. This had to be chiselled away before the artery could be ligated. Whilst the artery was being ligated the patient's respiration ceased. Artificial respiration was adopted and her head dropped and then she began to breathe again fairly quickly. The operation was proceeded with and no more trouble met with until Meckel's cave was opened and the root exposed. The patient then ceased breathing, all hæmorrhage stopped, she lost her colour which up till then had been good, and became an ashy grey colour. The pupils dilated to the limit, no heart beats could be felt or heard; the anæsthetist and I both thought her dead. It was decided to open the abdomen and to do cardiac massage and so as rapidly as possible and without aseptic precautions the abdomen was opened through the left rectus and the heart palpated throught the left side of the diaphragm. No cardiac impulse could be felt and so it was massaged against the chest wall. After a few moments it gave a feeble beat and then commenced to beat regularly. Respiration commenced and the anæsthetist said that the operation so near its close might be finished. As rapidly as possible the already exposed posterior root was divided with an Adson guillotine and the skull and abdominal wounds closed.

For the first forty-eight hours convalescence was extremely stormy and it was thought that she would go as other cardiac massage patients have gone some time after the operation without regaining consciousness. She threw herself about the bed so violently that to prevent her restlessness from causing her any injury she had to be shackled to the bed. For the whole of this time the only rest she obtained, was when she was under the influence of morphine. After forty-eight hours of this she regained consciousness and from then onward her convalescence was uneventful, but for the appearance of those herpetic patches in the mouth that occasionally mark cases of interference with the posterior root.

She left hospital in twenty-one days and has remained well. The abdominal wound, made without any aseptic precautions, healed by first intention.

LEFT HEMIPLEGIA (CEREBRAL PALSY).[1]

By G. C. Willcocks, M.C., O.B.E., M.B., Ch.M. (Sydney), M.R.C.P. (London),
Honorary Assistant Physician, Sydney Hospital.

The patient, a girl, aged eight years, came to the out-patient department, Sydney Hospital, with a history of wasting of the left hand of one month's duration. The child was said to have been fretful one night and had suffered from incontinence of micturition. The next day this wasting was noticed. There had been no previous illnesses. The mother stated that the child had been born after a breech presentation and had weighed 5·4 kilograms (twelve pounds). No instruments had been used. No illness had supervened after birth.

On examination it can be seen that the left arm is shorter and the left hand smaller than the right. The hand is rather glossy, the arm somewhat rigid. The gait is characterized by slight stiffness in the left leg which is spastic. The tongue is deviated slightly to the left. The reflexes are not altered; ankle clonus cannot be obtained perhaps on account of the rigidity of the ankle. Plantar response is indefinite. No sensory changes have

[1] The patient described herein was shown at a meeting of the New South Wales Branch of the British Medical Association on August 12, 1926.

been found. The child is mentally very bright, quiet, unemotional and there is no history of fits. There is a tremor of the left hand, increased on movement. The signs are those of an upper motor neurone lesion, affecting the tongue, left arm and leg. The smallness of the left arm indicates a lesion of probably some years' standing.

The diagnosis lies between polioencephalitis, cerebral neoplasm and cerebral palsy of childhood. There is no history of headaches and no vomiting and there is no optic neuritis. No history suggesting polioencephalitis can be obtained, hence the probable diagnosis is infantile hemiplegia due to some lesion (hæmorrhage) occurring at birth or prenatal maldevelopment. The result of the Wassermann test is "doubtful."

If this diagnosis is correct the condition should improve and mental impairment should not become evident, but the left arm and hand are unlikely to become normal in size or in usefulness. Reeducation may help to maintain the function of the left side. The mother says that exercises have improved the parts, even in two weeks.

It is not unusual for parents to fail to observe a defective function following infantile paralysis until to the seventh or eighth year.

The condition is unusual in that there is no history of defective mentality or of fits. The latter may still develope.

PRIAPISM: A FIRST SYMPTOM IN MYELOID LEUCHÆMIA.

By A. E. Brown, M.B., Ch.B. (Cantab.), M.R.C.S. (Eng.), L.R.C.P. (Lon.),

AND

K. McKeddie Doig, M.B., Ch.B. (Melbourne),

Colac, Victoria.

The patient was referred to us on June 10, 1926, by Dr. J. Scott Paton, of Lismore, with the following history:

The patient was a man of thirty-six years, suffering from priapism, which had lasted twenty-four hours. The penis was very fully erected and was giving intense pain both at the tip and towards the root. During the past six months he had had four or five previous attacks of priapism which, however, disappeared in a couple of hours or less. Beyond this he had been quite well and had carried on his work as a motor mechanic uninterruptedly. He had meningitis after going into camp in 1915, but recovered from this and went to the war, where he served with an infantry battalion. There have been no other illnesses of note and he denies venereal disease. He is married and has one child.

On examination he is a dark, thin man, who apart from the suffering caused by the priapism does not look ill. His colour is very good and he is quite bright and alert mentally. Heart and lungs appear normal. On examining the abdomen the spleen was found to be exceedingly enlarged, the lower border being only about five centimetres (two inches) above the pubic symphysis and the notch was felt distinctly in its sharp edge at the level of the umbilicus. Palpation of the spleen was not painful and he had not noticed any pain in the abdomen. The liver was not enlarged. The penis was very fully erected and tender to the touch. So painful was the condition of the penis, that sedatives had to be administered frequently during the ten days he was under our care and owing to the pain he had to adopt the recumbent attitude throughout. Heat and cold were were tried as applications and though neither had much effect in alleviating the condition, he seemed to prefer the hot applications to the cold ones. As time went on he began to experience some difficulty in micturition. The urine contained a little albumin.

The knee jerks were equal and active and the plantar reflexes were flexor in type.

Examination of the blood gave the following result:

Red cells 4,000,000 per cubic millimetre.
Hæmoglobin 68%.
White cells 499,200 per cubic millimetre.

Differential count:

Polymerphonuclear cells 34·2%.
Myelocytes 58·8%.
Transitional cells 3·2%.
Large mononuclear cells 0·4%.
Lymphocytes 1·4%.

Some polychromatophilia and an occasional nucleated red cell were seen.

Ordway and Gorham in the "Oxford Medicine" writing on priapism as a symptom of myeloid leuchæmia, state that:

> Contrary to the general impression, the percentage of cases showing this symptom is small. It is occasionally an early manifestation, but most commonly a late one . . . The cause is a myelocytic thrombosis or infiltration of the *corpora cavernosa*. The duration may be for a few weeks to seven months.

The interesting feature of this man's condition are firstly the priapism and secondly that in spite of the profound blood changes and the enormous enlargement of his spleen, the man felt quite well and carried out the hard manual labour of his occupation without effort, until the priapism forced him to seek medical aid.

Reviews.

A HANDBOOK FOR LABORATORY WORKERS IN PUBLIC HEALTH.

The eighth edition of "Public Health Laboratory Work" by Professor Kenwood is the complementary *vade mecum* of the well known standard textbook on hygiene by Parkes and Kenwood.[1]

The book is specially designed to cover the chemical branch of public health laboratory work as prescribed for the various courses for the Diploma in Public Health, the necessary hallmark of those who aspire to take up the speciality of public health.

The methods of collecting, examining, analysing and reporting on samples of water, sewage and sewage effluents, air, food and disinfectants are described so that the student can not only follow the various processes in the laboratory, but also interpret the results.

The reduction of sulphates present in sewage seaweed to sulphites, eventually liberating sulphuretted hydrogen, explains the occasional nuisance on the shores of some of our tidal rivers such as the Parramatta.

It is interesting to read that only one-sixth to one-eighth of the cow-dung which finds its way into milk is recoverable (as dirt) from milk by centrifugalization. As a rough household standard half a pint of milk placed in an ordinary tumbler should not throw any visible sediment in two hours.

The only satisfactory standards as to safety for use it is claimed are those based upon bacterial counts, as dirt may be partially removed by trade filtration.

The book which is frequently and well illustrated, can be commended to the laboratory worker and especially to those preparing for administrative and other positions in the ever widening sphere of preventive medicine.

THERAPEUTICS AND MATERIA MEDICA.

The fourteenth edition of Potter's "Therapeutics, Materia Medica and Pharmacy," revised by R. J. E. Scott, is to hand.[2] The first edition of this work was published in

[1] "Public Health Laboratory Work (Chemistry)," by Henry R. Kenwood, C.M.G., M.B., F.R.S. (Edinburgh), D.P.H., F.C.S.: Eighth Edition; 1925. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 381, with illustrations. Price: 12s. 6d. net.

[2] "Therapeutics, Materia Medica and Pharmacy," by Samuel O. L. Potter, A.M., M.D., M.R.C.P. (London); Fourteenth Edition, Revised by R. J. E. Scott, M.A., B.C.L., M.D.; 1926. Philadelphia: P. Blakiston's Son & Company. Royal 8vo., pp. 986.

1887 and the second edition with seven hundred and sixty-six pages in 1890. The present edition has been much enlarged and revised. New material has been added. Many statements have been modified and portions of the earlier editions deleted. This is a comprehensive volume containing a mass of statements concerning the therapeutic efficiency of hosts of vegetable and synthetic drugs without any effort to consider critically the genuineness of such claims. As curiosities of medical practice many of these drugs are interesting, but the book, as a guide to the efficient treatment of disease is not enhanced in value by their inclusion. The various parts of the book are devoted to empirical and rational therapeutics, constituents of organic drugs, methods of administration of drugs, dosage of medicine, *materia medica* (in which chapters the drugs are alphabetically arranged, irrespective of their pharmacological affinities—a very faulty method indeed), special therapeutics including poisoning and antidotes, pharmacy and other matters. As regards the writing of prescriptions, stress is rightly laid upon the necessity of not using ambiguous abbreviations which may be very dangerous. Potter's table of incompatibles is only generally true and quite inaccurate in several particular instances. Chaulmoogra oil and its derivatives are only very briefly mentioned in the treatment of leprosy and without any enthusiasm. Chalk is extolled as a basis for dentifrices. But chalk, being alkaline, may interfere with the salivary secretion and so be harmful. There is reason to think that alkaline dentifrices are injurious. In one place amyl nitrite is advised for dangerous symptoms occurring during chloroform anæsthesia. Elsewhere, however, amyl nitrite is correctly stated to be a dangerous remedy on account of the fall of blood pressure which it induces, the blood pressure being already unduly low from the chloroform itself. The treatment of pulmonary tuberculosis by gold salts is not mentioned. It is, however, truly stated that for alcoholism gold salts act only by means of suggestion and may produce dangerous cardiac failure. A number of animal extracts are described and their efficacy more or less taken for granted, without being scientifically examined. The "Insulin" treatment of diabetes is very scantily described. No mention is made of the treatment of pertussis by hypodermic injections of ether. Ergot is advocated for hæmoptysis; advice which we trust nobody will follow. Mistakes in spelling occur, such as "Licoperdon" for "Lycoperdon." Some good parts of the volume deal well with the toxicology of tea; the cocaine habit; quinidine; convallaria in cardiac disease; carbon dioxide snow; electro-therapeutics and vaccines and sera. Probably the worst parts of the whole volume deal with snake bite. Antivenene is absolutely specific and the apparent instances of a particular antivenom serum being efficacious against the bite of a different venomous snake have been found on subsequent investigation to be fallacious. Of this the author does not seem to be fully cognisant, although he refers to C. J. Martin's investigations on Australian venoms. It is almost laughable to read in this book that "*Agave virginica* is known in South Carolina by the name of rattlesnake's master . . . *Echinacea* (Black Sampson) is used with invariable success." In one place we read: "Alcohol, 1—4 oz. of whisky every fifteen minutes, as a stimulant in acute depression threatening a fatal issue, but is not antidotal as generally believed." Again: "Alcohol freely in snake bite, as a stimulant is most important. This belief is false; in a person badly poisoned by snake venom, the medullary centres are depressed and threatened with paralysis, and large doses of alcohol increase this tendency and may hasten death." Again we read: "Strychnine, hypodermically has proved eminently efficient in numerous case (Baron von Muller)." It was not Baron Ferdinand von Mueller at all who introduced this treatment but Dr. A. Mueller, of Victoria, who used heroic doses. Strychnine in no sense can be considered an antidote. The late Dr. Huxtable's statistics showed a mortality four times as great by strychnine treatment as without it and we have the unhappy suspicion that some of the fatalities after snake bite were caused by strychnine poisoning.

Perhaps the gem of the whole book is the instruction to treat spider bite or scorpion sting: "If in the wilds, burn with fire or gunpowder."

Plants indigenous to Australia receive considerable mention. For instance the species of *Duboisia*, yielding duboisine (a mixture of alkaloids) and nicotine, the latter being the aboriginal narcotic drug "Pituri," are mentioned; the former is rather extensively discussed. Abrus or jequirity is also indigenous to Australia, but we had hoped that the barbarous treatment of trachoma by this method had become obsolete. Cajaput has a rather wide distribution, extending plentifully over northern and eastern Australia; the habitat assigned in the book—Molucca Islands—is altogether too restricted. Eucalyptus oil comes in for a good deal of mention. It has not much honour in its own native habitat—Australia. It is correctly pointed out that cineol, cajuputol and eucalyptol are one and the same substance. Other indigenous Australian plants mentioned are Azedarach (our white cedar) and Kamala—both anthelmintics; the former for round worms and the latter which at one time was in the British Pharmacopœia, for *Tænia* species. *Euphorbia pilulifera* is the Queensland asthma herb, largely grown in the United States. It has a great vogue for bronchitis and asthma which is not accepted by most authorities. Kava-kava, largely used as an intoxicating drink by the Pacific islanders, is indigenous also to New Guinea. Therapeutically it is of value in cystitis and gonorrhœa. Australian kino (from species of eucalyptus) receives mention.

BACTERIOLOGICAL STUDIES.

Prefaced by two lines from Sir Ronald Ross's occasional verse "Tho' we may never reach the Peak, God gave this great commandment 'Seek,'" the Pickett-Thomson Research Laboratory has published another set of laboratory studies, almost entirely the work of Dr. David Thomson.[1] One of his objectives is to record photographically the whole range of bacteria and protozoa. He is an enthusiastic bacteriologist and is particularly "faddy" on the subject of culture media. Thomson's gonococcus medium was well known towards the end of the war and he has continued along this line. If heated in the presence of acid or alkali, agar-agar will actively inhibit bacterial growth and therefore sterilization must be done at the neutral point. Prolonged heating must be avoided and he effects this by substitution of sedimentation for filtration. Ordinary beef-infusion-peptone-agar will not grow the gonococcus unless enriching substances such as blood derivatives be added, but the latter are unnecessary if ox testicle-infusion be substituted.

Some work has been done on the cultivation of minute but microscopically recognizable organisms such as the *Bacterium pneumosintes* of Olitsky and Gates. There is some evidence that this anaerobic organism which passes certain filters, has an ætiological relationship to true influenza. Thomson claims to have discovered a small group of allied organisms in the respiratory tract and gives excellent photographs of them. He has found that boiled contaminated blood usually enhances the growth of these and also of the better known hæmophilic bacteria such as Pfeiffer's bacillus. He gives descriptions and photographs also of the pneumococcus types and of other bacteria, but little is added to our knowledge. The author is the exponent of detoxicated vaccines and an essential part of the process is the solution in alkali of the bacterial bodies. Some are easily dissolved, others with great difficulty, but the task is made easier if the bacterium can be cut up or smashed. To accomplish this he has used much ingenuity and patience in designing pieces of apparatus. In one of these, called the Thomson-Macfie vaccine churn, emulsions of bacteria are ejected through slots in a centrifugal container (at the rate of a mile per minute) against knife edges. It does not appear that any method is as yet an unqualified success. Apparatus has also been designed for the disintegration of tissues, endocrine organs and calf lymph.

It may be inferred that this volume is a somewhat unorthodox research publication mainly of interest to bacteriologists and technicians in large institutes and proprietary laboratories. The printing and photomicrography are excellent.

[1] "Annals of the Pickett-Thomson Research Laboratory," Volume I.; 1925. London: Baillière, Tindall & Cox. Demy 4to., pp. 84, with illustrations. Price: 17s. net.

The Medical Journal of Australia

SATURDAY, OCTOBER 16, 1926.

The Medical Congress.

In less than four months the second session of the Australasian Medical Congress (British Medical Association will be opened. On Thursday, February 3, 1927, the medical profession in Dunedin will stretch out its hands to welcome its colleagues from other parts of the Dominion of New Zealand and from the Commonwealth of Australia. The scene will be set in the new Medical School of the Otago University, a fitting stage for so important a function. The hosts have bidden many to the feast, but up to the present only a few have responded to the invitation. They have laboured assiduously and well in order that the meeting may be fruitful in advancing the science of medicine and in sealing a bond of friendship and kinsmanship between medical practitioners in the Dominion and in the Commonwealth. History is in the making. It is true that there have been two sessions of medical congresses in New Zealand in the past. In 1896 the fourth session of the Intercolonial Medical Congress was held in Dunedin, with Dr. F. C. Batchelor as President; in 1914 the tenth session was held in Auckland under the presidency of Dr. A. C. Purchas. It is interesting to note that at the Auckland session Dr. J. Young, speaking on behalf of the Council of the New Zealand Branch of the British Medical Association, proposed that at all future Australasian Medical Congresses the members should be limited to members of the British Medical Association. As no notice of motion had been given, this expression of opinion remained for a time a wish. Later it was determined to wind up the Australasian Medical Congress and this was carried out at the eleventh and last session at Brisbane. As a result of this action of self-extinction, the Federal Committee of the British Medical Association in Australia was able to establish a new congress whose organization would have none of the defects of its predecessor. The Federal Committee evinced wisdom in inviting the New Zealand Branch to join with the six Branches in Australia in adopting the new congress in the place of the old. The Federal Committee has constitutionally no relationship with the New Zealand Branch, but it has maintained the same procedure toward the New Zealand Branch as it did toward the Victorian Branch and the New Zealand Branch has voluntarily accepted this procedure. The second session therefore means something more than did the two former meetings of congress in New Zealand. It means the consummation of the first attempt at collaboration on the part of the New Zealand Branch of the British Medical Association with the Branches in Australia. It means that the medical profession in Australia is desirous of following a common policy, of combining in the endeavour to raise the standard of medical practice and to add to the knowledge of medical problems with special reference to Australasian conditions. The desire to amalgamate the forces of the sister colonies dates back to the beginning of the Intercolonial Medical Congress in 1886; it was reemphasized at Dunedin ten years later, when Professor L. E. Barnett, the learned President of the session of 1927, initiated a debate on the desirability of establishing an intercolonial medical journal.

The task of planning and preparing a modern congress is never a light one, but when it is undertaken in a small university town, far removed from other seats of learning and from large centres of medical activity, it becomes very difficult. If, added to the handicaps of limited scientific resources there is the extra burden of a relatively small attendance, the work becomes doubly difficult. The Executive Committee has conducted its business with determination, adroitness and skill. Visits by prominent members to Australia have been planned at well chosen stages and those concerned have been consulted in regard to the building up of the scientific programme. No stone has been left unturned in the eagerness and earnestness of the Committee to make the second session a great occasion. The subjects to be discussed are of immediate interest to every medical practitioner in Australasia and to the

medical profession of the world. The messages to be delivered will be important messages; they will bear evidence that the old world does not monopolize originality of scientific thought nor ingenuity in approaching difficult medical problems. For these reasons it may be claimed that every medical practitioner whose interests are in the advance of medical knowledge, whose desire is to join hands and enter into friendly competition with his New Zealand *confrères*, and who believes in the proper recognition of well directed endeavour, should make a supreme effort to be present in Dunedin on February 3, 1927. No one will regret the decision to become a member of Congress. The journey to New Zealand, the incidental attractions of the session, the entertainment of our Dunedin colleagues, the novelty of a holiday combining work with play in delightful surroundings, all will contribute toward a reward for the time devoted to this event. It is the duty of all to contribute toward the success of the second session of the Australasian Medical Congress (British Medical Association). There must be listeners as well as speakers at every successful meeting.

Current Comment.

INSENSIBLE PERSPIRATION.

The human body is regarded by physiologists as an elaborate piece of machinery in which combustion is continually taking place. Food in the form of fluids and solids is the fuel which keeps the machinery going. Food is taken into the body, altered, assimilated and burned up in the tissues. The waste products are excreted by kidney and bowel and by the skin in the form of obvious sweat. The weight lost by excretion of waste products may be measured, but estimation of the visible waste does not conclude the whole matter. A loss of weight is continually occurring owing to invisible gaseous emanations in the form of carbon dioxide and water vapour, given off from the lungs and the skin. In the past many attempts have been made to measure this invisible loss. It is obvious that this must be a more or less difficult procedure. In the first place it is not easy to build scales which will measure variations of a fraction of a gramme in so heavy a mass as the human body. Even if this degree of accuracy is obtainable, there is the second consideration of associated factors to be taken into account. These include such questions as the ingestion of food (solids and fluids) in relation to the time of weighing the body, the passage of fæces and urine, the position assumed by the subject of the experiment and the recent exercising of the muscles of the body, the temperature and humidity of the surrounding air.

This whole question has recently been the subject of an interesting communication by Dr. F. G. Benedict and Dr. H. F. Root.[1] They trace in an entertaining fashion the work of various investigators on "*perspiratio insensibilis*" from the findings of Sanctorius in 1614 with his movable platform attached to a steelyard up to the work of the present day. Lining in 1740 held that changes in the season have a pronounced influence on insensible perspiration. Lavoisier and Seguin in 1790 reported that a man lost 0·583 to 1·7 gramme per hour or from 900 to 2,500 grammes in twenty-four hours by "cutaneous respiration and pulmonary transpiration and respiration." Colin found that his loss of weight from insensible perspiration varied from 28 to 35 grammes per hour when he was asleep, from 50 to 80 grammes when he was awake and that the figure was increased up to 200 grammes during excessive exercise. Lombard in 1906 reached "the acme of perfection" with his balance which was the combination of a true balance with a spring scale. He found in one set of experiments that the loss averaged forty grammes per hour and that in another set, when the temperature was considerably higher, the loss amounted to seventy grammes per hour. Dr. Benedict and Dr. Root refer to the work done in Australia by Osborne in 1910 with his "human balance." The subject was weighed at intervals of one hour in a wooden house. In the intervals he lay in the open air in a hammock about twenty yards distant from the house. They point out that because of large differences in temperature (both of the dry and wet bulb) the movement of the air and the variation of clothing worn large differences of weight were recorded. The loss for an hour varied from 362 to 44 grammes. The loss of weight in one experiment in which the subject chopped wood, was 840 grammes in one hour.

Some of the later investigators have studied the insensible perspiration in its relation to metabolism. Isenschmid in 1918 pointed out that the insensible perspiration is equal to the weight of the water given off, *plus* the weight of the carbon dioxide given off, *minus* the weight of the oxygen consumed. From the diet the amount of carbon dioxide produced and the oxygen required to oxidize the food can be computed. Hence, if the subject is in metabolic equilibrium, it is possible to estimate the insensible water loss. Meyer has studied the insensible perspiration in infants and has concluded that under conditions of repose and in an atmospheric temperature of about 18° C. the hourly insensible loss is approximately forty centigrammes per square decimetre of body surface whether the subject studied is a small animal like a guinea pig, an infant or an adult man. He believes that the in-

[1] *Archives of Internal Medicine*, July 15, 1926.

sensible perspiration obeys a law comparable to that which governs the expenditure of heat and that by its study certain clinical applications may be found. Schmitt found that the insensible perspiration after feeding on protein was about twice as great as that following the ingestion of carbohydrate. This is interesting when it is remembered that the increase in metabolism is much greater after protein than after carbohydrate feeding.

The work of Dr. Benedict and Dr. Root was undertaken because they realized that there is sufficient correlation between the insensible loss and the metabolism to make the measurement of the insensible loss of direct practical value. They aimed at increasing the sensitivity of the measurements and shortening the period of observation. Two methods of estimating the insensible perspiration were used. The first consisted in measurements by means of platform or "silk scales." These were sensitive to variations of ten grammes. They also used a sensitive balance capable of weighing one hundred kilograms and of showing variations of 0·1 gramme. It is not necessary to describe this balance in detail. Weighings were carried out with the sensitive balance over ten or fifteen minutes and up to twelve measurements were secured. Each of these served as a control on the remainder. When the platform scales were used the period of measurement was lengthened to cover several hours in order to render the experimental error less significant. It was found that body size, the ingestion of food and exercise had a pronounced influence on the insensible loss. In view of the fact that these considerations influenced the general metabolism, Dr. Benedict and Dr. Root claim further evidence of the relationship between the insensible loss and the metabolism.

They then turned their attention to the investigation of pathological conditions. Patients suffering from hyperthyreoidism were found to have a large insensible loss and patients suffering from diabetes were found to have a small loss. The metabolism in the former condition is high and in the latter low. A relatively large number of both types of patients was studied and the loss in weight was measured just before the metabolism was determined. The results of these measurements are plotted in a curve. The insensible perspiration, expressed in grammes per hour, is represented in the ordinate and the heat production for twenty-four hours, as computed from the oxygen consumption, in the abscissa. The general trend of points is represented by a straight line. They conclude that there is a relationship between the insensible perspiration and the simultaneously measured metabolism and that this relationship is sufficiently close to permit the use of the insensible perspiration as a general index of the probable metabolism of a subject. At the same time it is obvious that this method cannot replace the carefully recorded basal metabolism measurement made under standard conditions. Dr. Benedict and Dr. Root point out that the error in the prediction of the metabolism from the insensible perspiration has not yet been calculated. They hope that with more careful attention to control, to the environmental temperature and other details many deviations will be eliminated. It is necessary to remember that estimations of the basal metabolism are accompanied by an experimental error. Readers are referred to a paper by Dr. F. S. Hansman published in this journal on August 22, 1925, and to the discussion which followed the reading of that paper. Error may be made in the estimation of the surface area of the body, in the volumes of the oxygen and carbon dioxide of the sample of air, it may be doubtful whether the patient is actually at rest and the personal equation of the experimenter must be considered. At the same time no one will deny that when a careful estimation of the basal metabolism is made by an experienced investigator, using the tables of Du Bois, a result suitable for clinical use will be obtained. Competent observers have expressed the opinion that even a carefully estimated basal metabolic rate should not be accepted as sole evidence that a certain clinical condition is present. This is an eminently safe attitude to adopt in regard to most clinical tests. When one is used to check the other, better results are obtained. This is well seen in connexion with the insensible loss in weight, discussed by Dr. Benedict and Dr. Root, in several cases described by them. It must suffice to quote one of these. A female patient, aged fifty years, was suffering from nervous symptoms, regarded by a competent clinician as being due to hyperthyreoidism. Estimation of her basal metabolic rate gave a figure 38% above what is regarded as normal by the Harris-Benedict standard. The insensible perspiration loss was 32·7 grammes an hour. According to the work described by Dr. Benedict and Dr. Root this figure was not compatible with a diagnosis of hyperthyreoidism. Several subsequent estimations of the basal metabolic rate were made and it was shown that the previous high figure was due to nervousness on the part of the patient.

Further elaboration of this work will probably prove that it has a clinical field of usefulness. A balance such as described by Dr. Benedict and Dr. Root could be installed in a large institution without prohibitive expense.

THE MEDICAL DIRECTORY.

Circulars containing forms to be filled in for the compilation of the Medical Directory have been addressed to every medical practitioner in Australasia. Those who did not reply to the first invitation, received a second circular. There are still many who have not entered the details required on the forms and returned them to The Printing House, Seamer Street, Glebe, New South Wales. The form has been reproduced in several past issues and will be found in the Advertiser. Members who have not already sent in their forms, are earnestly requested to tear out the page, to fill in the necessary details and to transmit it to this address as soon as possible.

Abstracts from Current Medical Literature.

MEDICINE.

Prophylactic Serum Against Measles.

R. KOCHMANN (*Deutsche Medizinische Wochenschrift*, April 2, 1926) gives his experiences with the prophylactic measles serum of Degkwitz. He tried to arrest an outbreak of measles in a hospital, but was unsuccessful. Nineteen children were treated, but only six escaped measles. The prevention could not definitely be classified as successful because several had had measles a short time previously, one was too young and another died during the incubation period. Two children died during the course of the infection. Of the eleven patients who contracted measles five ran a normal course, the fever lasting on an average for seven days. Six had bronchopneumonia and *otitis media* as complications. This disproved the contention of Degwitz that the serum obviated complications. The dose used was ten cubic centimetres and a third of the patients suffered from serum sickness. In fact, these patients suffered more than the normal untreated patients suffered.

Dyspepsia.

E. P. POULTON (*Post-Graduate Medical Journal*, May, 1926) after detailing the symptoms of the disease states that the proper treatment of dyspepsia is based on the fact that protein foods require and call forth a highly acid gastric juice for their digestion; fats when mixed with other foods cause a film to form over the gastric contents, leading to diminished secretion of hydrochloric acid, a regurgitation of bile and the retention of food for lengthy periods in the stomach. Fat and protein foods, therefore, should be taken as far as possible at separate meals. The plan of taking small quantities of food at frequent intervals is a good one. As regards fluids the "water of hydration," intended to replace losses through the sweat, urine *et cetera*, should be taken half an hour before meals. But a small quantity of fluid is necessary to reduce a dry meal to the proper moist consistency suitable for the action of the gastric juice and this should be taken with the food. All beverages should be taken warm; water, weak tea, cocoa or milk are suitable. A drink containing diastase (a decoction of malted barley which has been placed in a saucepan of boiling water for ten minutes) is a useful aid to digestion. The carbonates—the so-called alkalis—are valuable drugs. They act by liberating carbon dioxide in the stomach, thus stimulating contraction and preventing the stretching of its muscle fibres. The carbonates generally used are those of magnesium or sodium in doses of 1·8 grammes (thirty grains). The expensive salt of bismuth is unnecessary. In cases of dyspepsia in which the hydrochloric acid is diminished, the acid may with advantage be given by the mouth, especially it is found to patients suffering from rosacea. Poulton is very sceptical as to the value of various vegetable bitters frequently prescribed in dyspepsia.

Treatment of Diarrhœa.

R. D. RUDOLF (*Canadian Medical Association Journal*, May, 1926) classifies diarrhœa as being nervous, purgative and organic in origin. The nervous type in which there are no organic changes in the bowel (for example "examination diarrhœa," lientery *et cetera*), requires the use of nerve sedatives and also of belladonna to subdue colic if it should be present which is not usual. *Liquor arsenicalis* for some strange reason appears to be of benefit in this disorder. In the purgative variety of diarrhœa no organic changes are to be noted in the bowel. Some substance has been swallowed or elaborated by the stomach or bowel and acts as a purge. The diarrhœa is accompanied by colic and there is fairly complete relief in the intervals between the attacks. Medicinal purgatives act in this way and *achylia gastrica* may produce diarrhœa of this kind which is typical of an affection of the middle portion of the intestine. Bacterial decomposition of the bowel contents and chilling of the abdomen are common causes. The irritant, if suspected, may be removed by purgatives of which castor oil is the best for the purpose. Opium may be combined with it, if colic is present. Rest in bed with heat to the abdomen is essential in severe cases. The diet should be cut down to a minimum or omitted entirely. Feeding may recommence after a suitable interval with boiled milk foods which have an astringent effect. In diarrhœa due to organic causes the pathological changes in the lining membrane of the bowel cause increased secretion and peristalsis. In typhoid fever and in intestinal tuberculosis the lesions occur mainly in the mid-portion of the intestine; in cases of dysentery and colitis the lower bowel is affected. Dysentery admits of specific therapy, but in other forms of diarrhœa of organic origin treatment is purely symptomatic. Purgatives have a limited use, since the infection is lodged in the mucous membrane beyond their reach. For the commoner types of diarrhœa bismuth, chalk and opium may be used. In very acute watery diarrhœa kaolin is useful, sometimes even in Asiatic cholera. In the more chronic cases, tannic acid (as "Tannalbin" or "Tannigen") may be employed. A large number of intestinal antiseptics have been tried for chronic diarrhœa but it seems difficult to believe that these drugs would kill pathogenic germs and spare the harmless or beneficent. It is claimed that certain antiseptics *in vitro* kill streptococci and leave colon bacilli unharmed. While Garrod has stated that these antiseptics do not appreciably reduce the numbers of living aerobic organisms in the fæces, there is still some clinical evidence of their value. β naphthol has enjoyed a long popularity and may be tried in the more chronic type of case. "Dimol" now largely used is said to be thirty-five times as toxic as phenol to *Bacillus typhosus* and yet non-toxic to its host. Lavage with antiseptic or astringent solutions is preeminently suitable treatment for diarrhœa affecting the lower portion of the intestine and in very obstinate cases irrigation of the whole colon may be successfully undertaken after the operation of appendicostomy.

Blood Changes in Pertussis.

J. C. REGAN AND A. TOLSTOUHOV (*Journal of the American Medical Association*, April 10, 1926) have performed six hundred and eighty-two chemical analyses of the blood of patients with pertussis. They find very constantly a diminution of the total inorganic phosphorus and a lowering of the hydrogen ion concentration of the blood. These changes are always well established in the first week of the disease and both run a more or less parallel course. In patients treated with alkalis the inorganic phosphorus rises steadily from the third week; in those untreated no rise occurs until the sixth week. The same phenomenon is noted in the case of the pH value. The rise and fall of the inorganic phosphorus concentration is unconnected with age and is unassociated with the presence of rickets. The calcium content of the blood is not affected in any constant manner. The above blood changes are characteristic of an uncompensated acidosis, due to increased concentration of carbon dioxide in the blood and is manifested by vomiting, convulsions and the typical paroxysms. The vomiting seen in the course of the disease may be considered as an effort of the body to eliminate acid. Sodium bicarbonate, calcium carbonate and magnesium oxide will abort the disease. if administered early. The authors discovered the use of alkalis because of the clinical effect of the Sippy treatment on a patient with a gastric ulcer who suffered from pertussis. Alkalis produce a rapid rise of the inorganic phosphorus and a change in the pH of the blood with cure supervening in a relatively short period.

Pernicious Anæmia.

K. FABER (*Annals of Clinical Medicine*, April, 1926) discusses the intestinal origin of pernicious anæmia. Faber and Bloch have shown that there is no atrophy of the intestine in that disease, but it is known that typical pernicious anæmia occurred frequently when bothriocephalus is present in the intestine and that it is usually cured when the worm was expelled. Also typical pernicious anæmia has been recorded several times in association with stricture of the small intestine and in patients with sprue. *Achylia*

gastrica often precedes pernicious anæmia for many years, but since achylia often occurs without pernicious anæmia, it can not be the direct cause of that condition; in achylia the stomach's disinfecting power is in abeyance and the bacterial flora of the large intestine is continued right up to the duodenum, whereas normally the bacterial flora of the small intestine is scanty and homogenous. Streptococci, *Bacillus coli* and other organisms are found in duodenum and small intestine in achylia. As a result of the deficient acid proteins are not broken down completely and abnormal products flood the duodenum and jejunum. Seyderhelm prepared a protein from bothriocephalus which on injection into animals caused a true hæmolytic anæmia of the pernicious type and Nyfeldt produced similar results with extracts of cultures of *Bacillus coli*, enterococci and streptococci. The presence of these organisms in the small intestine where absorption is greatest may therefore be the cause of pernicious anæmia. A blood picture of the pernicious type in cancer of the stomach can be explained in a similar way. Periods of remission in pernicious anæmia may be due to a process of temporary desensitization, the result of the absorption of the toxins in the duodenum and jejunum. In pregnancy and sepsis the causative toxin is found not in the intestine but elsewhere.

A. C. REED and H. A. WYCKOFF (*American Journal of Tropical Medicine*, May, 1926) discuss the common picture of sprue, pernicious anæmia and combined degeneration of the spinal cord. These three conditions are all characterized by similar changes in the blood, gastro-intestinal system and spinal cord in many instances. In sprue stomatitis, gastro-intestinal atrophy, diarrhœa, progressive anæmia (often of the pernicious type) and nervous disturbances such as mental changes, anæsthesia, paræsthesia and flexor contractions occur; *achylia gastrica* is often noted. In pernicious anæmia the signs may be very similar. In subacute combined degeneration gastro-intestinal disturbances are not prominent, but gastro-enteritis, jaundice, diarrhœa and gastric irritability have been noted. Otherwise the signs closely resemble those of pernicious anæmia and of some cases of sprue. The view is advanced that all three conditions are due to toxin originating in the gastro-intestinal tract, not a single toxin but a related group possibly produced by a variety of causes. In the treatment of sprue diet, transfusions, change of climate, eradication of focal infection and symptomatic remedies are advised. Four cases of sprue are quoted to illustrate the points mentioned above. Two of the patients had symptoms and signs referable to lesions of the spinal cord—numbness, paræsthesia, anæsthesia and absence of the patellar and Achilles jerks. Two other cases are quoted in which the symptoms and signs were such that the condition could not be truly classified as sprue, pernicious anæmia or subacute combined degeneration of the cord, though in one case the onset was very suggestive of sprue and the other was more closely related to pernicious anæmia or subacute combined degeneration. The similarity of symptoms and signs suggests a common cause. In the case of sprue there may be some additional endemic factor which is potent in districts where sprue is wont to occur.

Liver Function Tests.

J. ROUILLARD (*La Presse Médicale*, April 21, 1926) discusses some new methods of investigating hepatic function. The liver has many duties. It excretes pigments and bile salts, it takes part in the metabolism of carbohydrates, fats and proteins, it stores reserves of iron, it cooperates in temperature regulations and it eliminates toxic substances and products foreign to the organism. When the liver is diseased, the various functions are affected unequally. The alteration in the percentage of bile pigment in the serum has been studied most. Fouchet's method consists in transforming bilirubin to biliverdin and comparing the green colour with a prepared solution. Van den Bergh mixed one cubic centimetre of serum with two cubic centimetres of 96% alcohol, a precipitate formed, the solution was centrifuged, two cubic centimetres of supernatant fluid drawn off and mixed with 0·25 cubic centimetre of a reagent (sulphanilic acid solution, hydrochloric acid and an aqueous solution of sodium nitrite) the colour being compared with that of a solution of cobalt sulphate. Others consider that the mere colour of the serum forms a useful guide to the degree of bilirubinæmia. Three cubic centimetres of blood are drawn off and allowed to coagulate in the dark. They are centrifuged and the supernatant fluid is compared with a one in ten thousand solution of potassium bichromate. The icterus index is two to five in normal conditions, six to sixteen in mild biliary retention and over sixteen in jaundiced subjects. The method has faults, but is fairly reliable. It reveals latent icterus in pneumonia, exophthalmic goître, toxæmias of pregnancy, during arsenical treatment, after chloroform administration and in early stages of infective or toxic jaundice. Rosenthal's method of injecting phenoltetrachlorphthalein has been considered as too dangerous for common use. Phenoltetrabromphthalein sulphate of soda (5% solution) two milligrammes per kilogram body weight, injected into a vein yields certain information. Five cubic centimetres of blood are drawn off in half an hour, coagulated and centrifuged. The clear serum is placed in two tubes, to one is added two drops of 10% soda solution, to the other one drop of 5% hydrochloric acid. These solutions are compared with standard solutions of "Bromsulfaleine." In liver affections, carcinoma, catarrhal jaundice, cholecystitis *et cetera* high percentages are found. In normal subjects two percentages are found a few minutes after injection and on coloration in half an hour. Diiodotetrachlorfluorescin of the series of triphenylmethane (Bengal rose) is nontoxic. It is injected intravenously and blood is drawn off two, four and eight minutes later; this blood is centrifuged, the serum diluted and compared with a standard. A definite retention is noted in cirrhosis, cancer of the liver and obstructive jaundice, but not in hæmolytic jaundice. It appears that the colorimetric methods of examination give results comparable to those obtained by testing the bilirubin content of the serum, but not altogether parallel. The actual significance of none of the methods is fully understood and the clinical examination and history are of greater importance in diagnosis.

Vaccination Against Tuberculosis.

G. POIX (*La Presse Médicale*, June 19, 1926) discusses a method of vaccination against tuberculosis evolved by Calmette and Guérin after a study of twenty years. The tubercle bacillus was cultivated on potatoes cooked in glycerinated ox bile, 230 successive cultures were made in thirteen years and colonies were obtained which were harmless to all animals including anthropoid apes. These cultures, however, continued to secrete tuberculin and provoked the formation of antibodies when injected, as could be shown by the complement fixation test. The vaccine produced from these cultures (called B.C.G.—*Bacille bilié* Calmette-Guérin) protected young rabbits, guinea pigs, calves and apes from tuberculosis when ingested or injected into the veins; control animals were used in all cases. The animals inoculated with the vaccine in no case became affected by tuberculosis; rarely mild local reactions occurred. This vaccine was used only in very young animals in which no tuberculous infection had occurred and when the method was applied to man only new-born children were treated since the use of tuberculous vaccines in those already infected with tuberculosis was held to increase the sensitiveness to reinfection. More than five thousand children a few days old have been inoculated in France and a comparison of the mortality of these children with the uninoculated showed that the mortality from tuberculosis in the former was about 0·7% and in the latter about 25%. Only children in whom the von Pirquet cutaneous test yielded no reaction, were inoculated. The bacterial emulsion was administered by mouth before it was ten days old; after that date it contained too few living bacilli to be effective. Three doses were given on the third, fifth and seventh days after birth. Immunity occurred after some weeks. The duration of immunity was uncertain, in calves it was about two years. The vaccine was produced at the Pasteur Institute.

British Medical Association News.

SCIENTIFIC.

A meeting of the Victorian Branch of the British Medical Association was held in the Medical Society Hall, East Melbourne, on September 1, 1926, Dr. H. Douglas Stephens, the President, in the chair.

Cancer Research.

Sir George Syme read a paper entitled: "The Need for Propaganda by the Medical Profession with Regard to Cancer" (see page 503).

Dr. C. H. Kellaway read a paper entitled: "Recent Researches on Cancer" (see page 505).

Dr. J. Newman Morris said that he hoped that the members of the Branch would take Sir George Syme's address to heart and that the profession as a whole was about to abandon the policy of silence on matters of public interest. The prevention of malignant disease or the mitigation of its incidence was preeminent among questions upon which the medical profession should endeavour to instruct the public.

The Council of the Victorian Branch hoped to accomplish much more work of the nature of propaganda than had previously been attempted and with this object had instituted a Public Questions Committee of the Council. This committee would issue authoritative statements on medical matters of general public interest and would assist in the framing of propaganda amplifying that already issued by the Cancer Committee and the Health Association.

Personally he welcomed the departure from the conservative attitude and hoped that it would receive the support of the Branch.

Dr. H. Flecker commented upon Sir George Syme's criticism of general practitioners for their apparent neglect to apprise their patients of the significance of various signs and symptoms as indicating the possible onset of malignant disease.

He could not see how the general practitioner could adopt educative measures at present without violating the code of ethics. If a member of the British Medical Association attempted to give lectures on malignant disease in his own neighbourhood, he would no doubt be asked to furnish an explanation to the Ethical Committee. At the same time much might be accomplished by this means, as witness the records of the American Society for the Control of Cancer.

As he understood the ethical objection to public lectures on medical subjects by medical practitioners, it was that such a practice would enable many to air their own fads and would be utilized for ulterior purposes by such as sought advertisement and notoriety. Any such abuse could be counteracted by prescribing officially the exact nature of the information to be disseminated.

Dr. A. E. Taylor said that he was astounded by the implications of neglect on the part of general practitioners made by Sir George Syme in his address. In his experience general practitioners were constantly on the *qui vive* for indications of malignant disease in their patients and did not fail to give warning and urge thorough investigation when suggestive signs and symptoms appeared.

He had gained a strong clinical impression that cancer was infective in origin, but it appeared from Dr. Kellaway's paper that such a view of the nature of malignant disease had not been substantiated by experimental research.

Sir James Barrett said that the practical issue was the only concern of a meeting called with the object of promoting the spread of instruction calculated to minimize the incidence and diminish the prevailing high mortality from malignant disease in Australia. The cause of cancer was not known, but it was known that appropriate treatment instituted early in the course of the disease was very efficient.

It had been well said by the President of the Wisconsin University twelve years previously that if the genius and energy put into research were suppressed for ten years and the same directed towards the application of knowledge already gained, the world would be transformed. Of the three classes, those who pursued research work, those who disseminated the results of research and those who applied them, the general practitioner fell into the last category. All were equally important.

In the fight against the scourge of cancer nothing could be accomplished without the cooperation of the rank and file of the medical profession. The Health Association had prepared wall sheets and possessed cinematograph films designed to inform the public with regard to malignant disease. He asked that all who were in sympathy with the activities of the Cancer Committee and the Health Association would volunteer to give lectures and would avail themselves of the means of propaganda which the Health Association was anxious to place at their disposal.

Dr. A. Norman McArthur said that he welcomed the signs of activity directed towards the more effective control of malignant disease. Such efforts, though belated, were not to be regarded as too late.

His experience during the last four years of carcinoma of the uterus at Saint Vincent's Hospital was that in the majority of the women the growth had reached a stage at which it was inoperable. Some of the patients had been under medical observation, others had not. It was apparent that there was something wrong in the prevailing attitude towards possible incipient malignant disease.

He constantly impressed upon his students that any extraneous uterine discharge demanded careful and exhaustive investigation.

The age of the patient was not to be considered, for there were many records of uterine carcinoma developing early in life. The climacteric was the danger period and unfortunately many women regarded irregular bleedings at this time as quite natural. The general practitioner must have abundant opportunities for correcting this erroneous impression. It was an unfortunate fact also that no woman would believe in the possibility of cancer in the absence of pain, while actually when pain supervened the disease was entering upon its last dreadful phase.

It was very tragic that in the present days so many women who had been under medical observation, should miss the opportunity of surgical eradication of malignant growths early in their course.

With reference to carcinoma of the breast, Dr. McArthur said that diagnosis in the early stages was often impossible without the aid of the pathologist. The patient could always be persuaded to undergo the minor operation for the removal of a piece of the suspected tissue for microscopical examination. Within the last few months he had obtained a pathological report in ten instances in which the patient presented a lump in the breast. Nine of the specimens proved to be innocent, but the tenth, although on clinical grounds the probability was that it was benign, was shown to be malignant. To rely on clinical experience was to court tragedy.

Reverting to the subject of malignant disease of the uterus Dr. McArthur referred to the very encouraging progress in radium therapy and its possibilities in the treatment of neoplasms which had advanced too far for operative measures. He had been recently greatly impressed by some remarkable results achieved by irradiation. Much was due to Régaud, of Paris, in connexion with radiotherapy and in any propaganda work this aspect should not be overlooked. A ray of hope could be shown to women with inoperable uterine cancer and to extend it to them would help materially in gaining the public interest in a campaign against cancer.

Information on all points connected with cancer should be disseminated in an organized manner, the question of ways and means being for the Cancer Committee to arrange.

Mr. R. Hamilton Russell asked to whom it was proposed to address propaganda, the profession or the public? If to the public, the proposal occasioned him much misgivings; if to the profession, he could heartily support it.

Dr. McArthur's remarks expressed just what had been in his own mind and his own experience had been that there was a deplorable lack of promptness in instituting investigations directed towards the detection of malig-

nant disease. In his opinion nine-tenths of the delay was due to the medical man and no amount of propaganda to the public would obviate the other tenth.

Mr. Russell recalled Sir George Syme's presidential address on the occasion of the last session of Congress. To one passage in that admirable and eloquent address he was unable to subscribe and that was the advice given by Sir George that women should periodically examine their breasts for lumps. To his mind nothing could be worse and he regarded the suggestion as dangerous and undesirable. The person who became obsessed with a purely imaginary disease, was much worse off than the actual subject of that disease. He had only to mention the tortures undergone by syphilophobes.

He considered that propaganda addressed to the public would not touch the main source of the problem of malignant disease. People did not go to Chinese and herbalists through ignorance of the dreadful nature of cancer. They went because they knew friends and relatives, numbers of them, who had been "completely and permanently cured" of cancer by these irregular practitioners without operation. On the other hand they had plenty of other acquaintances who had been treated by surgeons and these seemed all to have undergone terrifying operations with a most meagre record of success as a result. Propaganda work directed towards the education of the public in its own interest appeared to be singularly unpromising.

Dr. J. A. Smeal said that he hoped that Mr. Russell was at fault. He had had eight years' experience of general practice and was convinced that the loss of time so much to be deplored was not the fault of the medical man. General practitioners as a whole were extremely careful not to overlook malignant disease which in fact was never out of their minds. Hitherto they had not received any assistance from the Branch as to the best means of advising the public in the matter and he would appreciate a concise leaflet which might be handed to patients.

Perhaps Mr. Russell did not appreciate the difficulties of the general practitioner. Owing largely to ignorance of the significance of serious symptoms patients frequently lost valuable time in following treatment prescribed by herbalists and other quacks. It was a common experience to see a patient with advanced malignant disease who had sought no advice other than that to be obtained from a chemist or a herbalist.

Further, people were often deliberately reticent. He had known nurses to conceal the fact that a lump which they knew demanded investigation, was present in the breast or elsewhere.

No harm could attend the distribution of information to the public and in his opinion it could do nothing but good. If a certain number of persons were made afraid, they could be easily disabused of baseless fears by consulting their doctors. It was by such consultations that many patients with malignant disease would be reached while the growth was in an early stage.

Sir George Syme, in reply to Dr. Flecker, said that there could be no ethical objection to personal communications. In regard to public utterances he pointed out that the very object of the institution of such bodies as the Public Questions Committee of the British Medical Association and the Cancer Propaganda Committee was that any public statement should be suitable and authoritative and given under the ægis of a recognized body. The question of ethics did not arise.

In his opinion propaganda should be addressed to both the medical profession and the public and on this point Mr. Russell and he must agree to differ.

He could not understand Mr. Russell's objection to his advice that women should periodically examine their breasts with a view to the detection of lumps. He had in mind many women affected by advanced carcinoma of the breast who had said that they had no idea there was anything the matter, in spite of the fact that a palpable lump must have been present for a long time. Cancer of the breast had been known to be discovered by physicians in the course of a routine examination of the chest. If a certain number of women were rendered doubtful about their breasts they could consult a medical man and have their fears dissipated or treatment commenced.

In any anticancer propaganda the public should be insistently informed of the danger of temporizing with herbalists and other quacks. The only possible way to deal with the menace of the quack was to educate the public, for unqualified practice could not be stopped by legislation.

Dr. Kelleway also replied briefly. He supported Sir George Syme in the contention that it was just as important to educate the public as to urge the early detection of cancer upon the medical profession.

MEDICO-POLITICAL.

A meeting of the Victorian Branch of the British Medical Association was held at the Medical Society Hall, East Melbourne, on September 1, 1926, Dr. H. Douglas Stephens, the President, in the chair.

Amendment of Rules of Ethical Procedure.

Dr. A. L. Kenny moved:

> That the following be added to Rule 7 with regard to Procedure of Inquiry into Complaints regarding Professional Conduct:
>
> The Council may at its discretion refer to the Ethical Subcommittee for consideration any newspaper article, paragraph or report which imputes to a member of the Association conduct detrimental to the honour of the profesison.

In outlining the reason underlying the proposed alteration of the Rule, Dr. Kenny explained that in all matters connected with ethics the Council was the judicial body and as such was precluded from laying any charges of unethical conduct. Members generally were reluctant to report ethical breaches and to do so appeared to be nobody's duty. The proposed addition would enable the Council to take cognizance of some matters which should be noticed.

In seconding the motion, Dr. A. V. M. Anderson said that it had been submitted only after mature consideration by the Council.

The motion was carried unanimously.

At a meeting of the Council of the South Australian Branch of the British Medical Association held on September 2, 1926, the following matters were considered.

Federal Committee Meeting.

Dr. H. S. Newland, C.B.E., D.S.O., and Dr. Bronte Smeaton gave an account of the business of the Federal Committee of the British Medical Association in Australia at its meeting on August 25, 1926. They reported that in connexion with the question of the introduction of uniform agreements with friendly society lodges, the Queensland Branch delegates had not received any instruction from the Branch, notwithstanding the fact that the matter had emanated from that Branch.

Appreciation was expressed at the work of the Lodge and Ethical Subcommittee in preparing the brief for the South Australian Branch. It was stated that the proposals contained therein were being utilized as the basis for further consideration by the Branches.

Post-Graduate Course.

On the motion of Dr. F. N. le Messurier, D.S.O., it was decided to appoint a subcommittee to organize a post-graduate course. The same members of the Council who had served on the subcommittee during the previous twelve months, were appointed.

Workmen's Compensation Act.

A letter was read from Dr. F. H. Beare in which he pointed out the difficulties experienced in connexion with the medical certificates issued under the *Workmen's Compensation Act*. It was resolved that Dr. Beare be advised that his construction of the views of the Council was the correct one and that the Council considered that the

instructions embodied in the printed slips for attachment to the medical certificate books were authorized.

British Medical Association Prize Essay.

The Honorary Secretary reported that a printed notice dealing with the establishment by the British Medical Association of a prize essay to the value of ten pounds for medical students of any university in the British Empire outside the United Kingdom had been received (see The Medical Journal of Australia, August 21, 1926, page 255). It was resolved to display the notice in the Adelaide University.

Section of Anæsthetics at Congress.

A letter from Dr. Gilbert Brown was read in which he suggested that as it was too late to form a section of anæsthetics at the second session of the Australasian Medical Congress (British Medical Association), Dunedin, 1927, the South Australian Branch delegates be instructed to propose the formation of such a section at future sessions. The Honorary Secretary was instructed to advise Dr. Brown that the matter had already been considered by the Federal Committee, but that as it was too late for anything to be done before the next session, the matter had been noted for future action.

Medical Association of Australia.

Dr. H. S. Newland, C.B.E., D.S.O., gave notice of motion as follows:

> That this Council takes the steps necessary to promote the formation of a medical association of Australia, of which the present Branches of the British Medical Association in Australia shall be Branches.

Medical Societies.

THE ALFRED HOSPITAL CLINICAL SOCIETY.

A meeting of the Alfred Hospital Clinical Society was held at the Alfred Hospital, Melbourne, on August 18, 1926. The meeting took the form of a series of clinical demonstrations by members of the honorary staff.

Renal Disease.

Dr. A. V. M. Anderson showed a series of patients who were suffering from renal disease.

The first was a man, aged forty-two years, who had recovered from two fits just prior to admission to hospital in August, 1926. In 1923 the patient had been admitted to hospital with œdema of sudden onset. No ascites had been present and the heart had not been enlarged. The urine had contained albumin, hyaline casts and leucocytes. The condition had improved with treatment and double decapsulation of the kidneys had been performed by Dr. Gordon Shaw. The patient had been fairly well since the operation. Œdema of the legs had occasionally been present and the abdomen had been tapped once on account of ascites. He had been on a restricted diet for some years, but latterly had been eating some meat.

The fit prior to admission to the Alfred Hospital had been of the nature of a clonic spasm. He had been unconscious for some minutes, no cyanosis had been present and the patient did not bite his tongue. On admission to the Alfred Hospital he had been drowsy, his tongue had been dry and furred and the urine had contained a few pus cells, granular *débris*, hyaline casts and an occasional motile organism. The day after admission a fit lasting for ten minutes had occurred. Venesection had been performed and saline solution had been given intravenously. The condition had slowly improved since then. Examination of the fundi revealed thickened arteries which nipped the veins, no other abnormality had been found. The blood urea was 207 milligrammes per hundred cubic centimetres. The urea concentration test gave a figure of 0·7% two hours and 0·6% five hours after the administration of urea.

Dr. Anderson's second patient was a boy, aged fourteen years, who had been admitted to hospital on July 28, 1926, with a history of having had bilious attacks not related to food for the previous five years. Three weeks prior to admission the patient had suffered from sore throat, swollen glands of the neck, vomiting and fever. Just prior to this his neck had been stiff for one week. For one and a half weeks before admission swelling of the arms, legs and face had been present. The patient had complained of backache and cough with some sputum. The day before admission a rash, not scarlatiniform, had been noticed on the chest and back. On admission the urine had contained albumin, blood, blood casts and granular casts, the specific gravity was 1012. The urea concentration test had yielded a figure of 0·8% at the end of the first hour and 1·4% at the end of the second hour. The blood urea had been 38 milligrammes, the systolic blood pressure 178 and the diastolic pressure 110 millimetres of mercury. The amount of urine passed in one day had been nine hundred cubic centimetres (thirty ounces). Examination on admission had revealed generalized œdema, a fading rash and no desquamation, carious teeth and chronic tonsillitis. The pulmonary second cardiac sound had been accentuated and reduplicated, the arteries were not thickened. Crepitations had been present at the bases of both lungs. Tenderness had been present in the costo-renal angle. Mild œdema of the left optic disc with some blurring had been found. Treatment had consisted of a citrate of potash mixture; compound powder of jalap, given every night; mustard plaster to the bases of the lungs; turpentine stupes to the loins; electric light baths twice a day and a carefully regulated diet. The œdematous signs had disappeared from the lungs in three days and the systolic and diastolic blood pressures had come down respectively to 132 and 92 millimetres of mercury. The blood pressure had remained at about the same level since then. The œdema had disappeared in a week. The urine occasionally contained a trace of albumin and some red blood corpuscles.

Dr. Anderson's third patient was a girl, aged twenty years, who had suffered from rheumatic fever twelve years previously and from diphtheria and scarlet fever nine years previously. She had been in the Austin Hospital for eight years. As a result of the rheumatic fever her joints had become affected and movements had become limited in both knees and hips. Later on the joints of the hands and the right wrist had become involved.

On admission it had been noted that the right hip and the right and left knees were semiflexed and that movements were limited. Treatment had been instituted by small doses of *Bacillus coli* vaccine, but no reaction had occurred. Adhesions had been broken down in the knees and massage and electrical stimulation had been used. The urine had contained a trace of albumin, the specific gravity had been 1014 and the patient had been placed on a restricted meat diet. The highest amount of albumin had been 6·5 milligrammes. The figures obtained as a result of the urea concentration test had been 1·4% at the end of the first hour and 1·5% at the end of the second hour. The urine contained pus cells and organisms. The patient was being treated by a citrate of potash mixture and improvement was noted. No septic focus had been discovered with the exception of spirillæ in the gums.

Dr. J. F. MacKeddie described the use of "Lipiodol" in the diagnosis of intraspinal and pulmonary lesions. He detailed the clinical histories of a number of assembled patients and demonstrated from a large number of radiograms and mural charts.

Artificial pneumothorax as a therapeutic measure in pulmonary tuberculosis was also discussed from the standpoints of indications, method of performance and the effects of the injection as observed by means of X rays.

Amnesia.

Dr. J. P. Major showed a male patient, aged forty years, a carpenter, who had been admitted on July 29, 1926, with a provisional diagnosis of amnesia. He had lost his memory for three days. Inquiry into his previous history had revealed that he suffered from "bronchial catarrh" five years previously and had been treated in Saint Vincent's Hospital. Nothing suggestive of *encephalitis lethargica*

could be discovered. The family history was unimportant. He had gone out one morning looking for work and had awakened four days later to find himself lying on the beach. He denied taking alcohol to excess. His memory had always been very poor. He had suffered from headaches and heaviness in the head and had noticed that on bending at work he sometimes saw double. On admission he did not consider that his left arm was weaker than usual. He had suffered, however, from occasional attacks of numbness in the left arm during the previous three or four years. On admission he had been found to be suffering from acidosis for which he was treated. No abnormality had been found in heart, lungs or abdomen. The central nervous system had appeared somewhat dull. The pupils had been equal and had reacted to light and accommodation. The reflexes had been active and equal, but the superficial abdominal reflexes were absent. The plantar reflex was flexor in type and no ankle clonus was present. Sensation was normal. Muscle power was good. The urine had been acid in reaction with a specific gravity of 1010, it contained albumin, but no sugar. The blood had reacted to the Wassermann test. The fundi were normal. The patient had been examined by Dr. Gamble and he had reported that the grip was less in the left than in the right hand, that the patient did not display any morbid mental symptoms and that all the muscles responded normally to faradism.

Epileptic Coma.

Dr. Major's second patient was a man, aged forty-five years, a gardener, who had been admitted on July 25, 1926, with a provisional diagnosis of epileptic coma. He complained of having had fits during the preceding nine months. The only thing of importance in the previous history was the fact that he had suffered from venereal disease thirty years before. The family history was unimportant. In describing his present illness the patient said that he had a peculiar sensation before the onset of a fit. He felt as if someone was trying to pull him down to the ground and was shaking him. He saw animals and figures before his eyes, the ceiling appeared to be falling on him and the floor to be coming up towards him. He became unconscious during the fits and used to bite his tongue. He had passed urine and fæces involuntarily. His vision was poor and at times he saw double. The action of his bowels was regular and he suffered from nocturnal frequency of micturition.

Examination revealed that his pupils were equal and reacted to light and accommodation. The heart was normal. No abnormality was detected in the lungs or abdomen. The cranial nerves were normal. The reflexes were very active, they were possibly slightly greater on the left side than on the right. The superficial abdominal reflexes were absent. The plantar reflex was flexor and there was no ankle clonus. The muscle power was good. The left hand grip was weaker than the right, but the patient was a right-handed man. The sense of position and of passive movement was impaired in the legs. Sensation of heat and cold was impaired on the left forearm. Tactile sensation was impaired on the nose. The vibration sense was good. The patient's power to discriminate between the points of a compass was impaired in the legs. The figures obtained as a result of the urea concentration test were 1·5% at the end of the first and 1·9% at the end of the second hour. The cerebro-spinal fluid was clear, but under increased pressure. It contained 0·04% globulin. No reaction had been obtained from the cerebro-spinal fluid in the Wassermann test, but the blood serum yielded a "++" reaction. The fundi were normal and the pupils reacted to light and accommodation, the patient suffered from left homonymous hemianopsia. Treatment was being carried out by the administration of iodide and bromide of potash and the use of mercurial inunctions. "Muthanol" and "Novarsenobenzol" were also being given.

Cardiac Lesions.

Dr. M. D. Silberberg showed a series of patients suffering from various cardiac lesions.

The first was a man, aged thirty-five years, who suffered from chronic heart block with an unusually rapid ventricular rate of forty-eight per minute. The condition had followed an acute septicæmic infection four years previously. The patient had suffered from several Stokes-Adams attacks, but had had none for months. His general condition was good. Dr. Silberberg pointed out that a large systolic bruit could be heard and that faint auricular sounds were audible. He also showed electrocardiograms of the condition.

The second patient was shown in order to illustrate an unusual type of venous bruit heard over the lower end of the sternum. The sound resembled wind whistling through the rigging of a ship. It was ascribed by Dr. Silberberg to slight rotation of the heart and torsion of the inferior *vena cava*. Dr. Silberberg regarded the bruit as of no significance. It was only slightly altered by the patient's change of position or respiration.

The third patient shown by Dr. Silberberg was a man, aged forty-two years, who suffered from mitral stenosis. He had suffered from rheumatic fever at the age of nine and in April, 1926, had contracted influenza. He had stayed at work and had become short of breath with palpitation and epigastric pain. When examined on June 2, 1926, he had manifested slight icterus, his liver was large and the pulse irregular, about 120 in the minute. The lower margin of the heart was 12·5 centimetres (five inches) from the middle line and a rough, slapping mitral first sound with a diastolic bruit was heard. Electrocardiographic examination had revealed impure flutter and aberrant beats. After the administration of digitalis the pulse had slowed to seventy-two beats in the minute and had still been irregular. Electrocardiographic examination had then revealed fibrillation. Quinidine treatment for two days restored a normal rhythm and the patient had returned to work. At the time of demonstration the rhythm was normal.

Dr. Silberberg's fourth patient was a woman, aged forty years, who suffered from mitral stenosis. Auricular fibrillation had occurred in 1922 and a normal rhythm had been restored for four years by quinidine. The heart's action had become irregular again in three months; the quinidine had not been repeated. Examination revealed an irregular pulse with a rate of 112 in the minute. The apex beat of the heart was situated 12·5 centimetres (five inches) from the middle line and the cardiac dulness on the right side extended one and a quarter centimetres (half an inch) from the border of the sternum. Systolic shock was present. There was a slapping first sound and a diastolic bruit. Dr. Silberberg showed electrocardiograms of the condition.

Dr. Silberberg's next patient was a woman, aged forty years, who suffered from mitral stenosis with a normal heart rhythm. She had suffered from acute rheumatism eighteen years previously and had undergone a surgical operation for a uterine condition and removal of the appendix seven years previously. Examination of the heart revealed a slight presystolic thrill and systolic shock. Pulmonary diastolic shock was also present. On auscultation a presystolic murmur and a slapping first sound were audible together with a diastolic bruit. The pulmonary sound was accentuated.

Chronic Gout.

Dr. G. A. Birnie showed a female patient, aged fifty-four years, in whom chronic gout was manifested by excessive deposition of sodium biurate. She had experienced her first attack of gout in the left great toe at the age of twenty-six years. No further attack had occurred for the next six years and then the second finger of the right hand became affected. Various acute attacks had supervened in the joints of both hands. During the last ten years the disease had become chronic in both the large and the small joints. Chalky material had first been discharged from the left elbow joint six years ago and since then the skin over many other joints had ulcerated as the result of biurate deposition.

Dr. Birnie said that the second finger of the right hand had been amputated two years and the second toe of the left foot eighteen months previously. Except for acute attacks of pain in the joints the patient felt very well. She stated that her father had suffered in a similar manner. It was curious that there were no gouty tophi in the

ears. Her systolic blood pressure was 150 millimetres of mercury and there did not appear to be any arterial thickening. The blood urea was forty milligrammes per hundred cubic centimetres. The urine did not contain any albumin. Acute attacks of pain every two to six months were always relieved by colchicum.

Urinary Calculi.

Mr. Fay Maclure, O.B.E., showed a large number of excellent skiagrams and pathological specimens of renal, vesical and prostatic calculi. He also demonstrated the method of use of the apparatus for the operation of litholapaxy.

Achlorhydria.

Dr. W. Summons, O.B.E., showed a series of patients who were suffering from achlorhydria.

The first was an unmarried woman, aged forty-six years, who complained of continuous epigastric pain of many years' duration which had no relationship to food. She vomited occasionally about fifteen minutes after the evening meal. The pain had always been relieved by *pulvis alkalinæ compositus*. Appendicectomy had been performed seven years previously and amenorrhœa had been present since the operation. The patient complained that the pain was worse since the operation. Her weight was constant. She complained of no other symptoms, except that she was "nervy," suffered from headaches and her bowels were constipated.

On abdominal examination tenderness had been present in the epigastrium and X ray examination revealed that the tone of the stomach was good, it was J-shaped and no filling defect was seen. The duodenal cap was well formed and no abnormality was detected. The patient's blood serum had not reacted to the Wassermann test.

The hæmoglobin value had been 73%, the leucocytes numbered 10,000 and the erythrocytes 4,600,000 per cubic millimetre. A fractional test meal had been carried out and a report of complete achlorhydria with an excess of mucus had been obtained. The urine was normal. The patient had been treated since early in May by dilute hydrochloric acid and a suitable diet. Her weight had increased, but the pain was still present.

The second patient was a man, aged sixty-one years, who had complained of pain in the abdomen of seven years' duration. The pain had occurred immediately after meals and lasted a variable time up to two hours. At first vomiting took place, but the patient had not vomited for four months. He had lost 6·3 kilograms (one stone) in weight. He had suffered from cough with sputum for some years. The family history was good. He was a tanner by trade and had been grinding bark for twenty-three years.

When the patient was first examined at the end of June, 1926, no abnormality had been found in the abdomen. The lungs were emphysematous and moist sounds were heard at both bases. No tubercle bacilli had been found in the sputum. A barium meal revealed no lesion of the stomach or duodenum, but X ray examination of the chest showed that fibrosis with accentuated hilum shadows was present. A fractional test meal revealed complete achlorhydria. No abnormality was found in the urine and the blood film was normal. The patient had been treated by the administration of dilute hydrochloric acid and had gained 0·9 kilogram (two pounds) in weight. He felt better, but said that he did not ever become hungry. He was suffering from a crop of boils on the neck.

The third patient was a man, a cook by occupation, who had been seen for the first time in October, 1925, complaining of pain in the stomach of three weeks' duration. The pain had been present constantly day and night and had been made worse by the ingestion of food. Vomiting had been present and relieved the pain. He had lost 3·6 kilograms (eight pounds) in weight. He complained that he had had a similar attack six months previously and that he had passed blood by the rectum and had vomited blood.

The patient had been admitted to hospital in November, 1925, and submitted to operation. A chronic gastric ulcer adherent to the pancreas had been found. The ulcer had merely been infolded as the condition of the patient would not allow of further treatment. Section of an enlarged gland removed at the time of operation manifested hæmorrhage and some capsular thickening. A fractional test meal had been undertaken in December, 1925, and a report received to the effect that no free hydrochloric acid was present and that active hydrochloric acid was 24% in a one hour specimen of gastric juice. The patient had been discharged from hospital on December 30, 1925, and had been comfortable until June 10, 1926. During this period he had not reported to hospital, but in June had complained of pain one hour after food. On X ray examination it was found that the stomach was high in position and that peristalsis was active, no ulcer niche or filling defect was seen. In July the patient still had epigastric pain and tenderness after treatment by alkalis. A fractional test meal in June had revealed a normal, acid curve, but fractional test meals on July 13 and July 30, 1926, had revealed complete achlorhydria. Bile had been present in all specimens taken at the test meal on July 30.

On August 2, 1926, the patient still had some epigastric discomfort. His appetite was poor. He vomited at night and food had no effect on the pain. No hæmatemesis or melæna were present. He was being treated by acids. He had gained weight at first, but had lost 0·45 kilogram (one pound) in a fortnight.

The next patient was a man, aged thirty-three years, a tanner, who complained of indigestion of five or six years' duration. A dragging pain unassociated with food had been present in the back of the left side of the chest for two months. The patient had vomited occasionally and constipation was present. On X ray examination the stomach was J-shaped, its tone was good, its size was normal and its peristalsis was feeble. No evidence of ulcer was found and the duodenal cap was normal. A fractional test meal revealed complete achlorhydria and the blood film was normal.

The last patient shown by Dr. Summons was a man, aged forty-nine years, who had been admitted in July, 1926, complaining of swelling in the joints of eight months' duration. He had suffered from asthma from the age of eighteen to that of forty-five. When a young man, he had been addicted to alcohol. Rheumatoid changes were present in the phalangeal joints, in both ankles and the left shoulder joint. No septic focus could be discovered and the patient was treated by large doses of *Bacillus coli* vaccine. A fractional test meal revealed complete achlorhydria.

Flail Foot.

Dr. John Kennedy showed a male patient, aged nineteen years, who had suffered from acute poliomyelitis in infancy. He had suffered from deformity and wasting of the left thigh and leg with flail foot. Otherwise the patient was healthy and well developed. Wasting of the thigh, of the leg and of the foot muscles was present and the ankle and mediotarsal joints had been deformed so that the weight was carried on the anterior and outer side of the foot; in other words a gross cavus deformity was present. On March 29, 1926, a lateral incision had been made and arthrodesis of the mediotarsal joint was performed. The plantar fascia had been cut and the foot put up in plaster in a position of dorsiflexion. On April 19, 1926, an incision had been made just lateral to the *tendo Achillis*. The cartilage of the talo-calcaneal and the talo-tibial joints had been removed in the shape of a wedge. The *tendo Achillis* had been sutured to the base of the bone at this site. The foot had been put in plaster with the talo-tibial joint in a state of inversion. The patient had been discharged on May 3, 1926, and on August 18, a special reinforcing boot was made for him.

Talipes Calcaneus.

Dr. Kennedy's second patient was a girl, aged fifteen years, who had suffered from anterior poliomyelitis at the age of three. Wasting of the right leg was present and involved especially the posterior group of tibial muscles. Shortening of the whole leg was present and this was equally distributed in both segments. A curved incision had been made on November 12, 1924, over the medial malleolus of the right foot and a wedge-shaped piece of bone had been removed from the tibia and calcaneus. In this way the proximal part of the ankle joint was removed.

The cut surfaces had been approximated to the periosteum by catgut. The *tendo Achillis* had been shortened by subcutaneous tenotomy.

The patient had been readmitted in July, 1926, with inversion of the foot. She was then walking on the outer surface of the foot. Operation had been undertaken on July 27, 1926, and scar tissue had been removed from both sides of the foot. Lateral cuneiform osteotomy was also performed and the foot put up in a position of slight eversion.

Ureteral Stricture.

Dr. Kennedy also showed a woman, aged twenty-six years, who had complained for twelve months of attacks of pain in the lower part of the abdomen. The attacks had been more severe on the left side and had lasted for from two to seven days. Associated with the attacks there had been increased frequency of micturition and occasional vomiting. General hypersensitiveness of the abdomen had been present, but this was most pronounced in the left iliac fossa. On May 28, 1926, cystoscopic examination had been carried out together with dilatation of the right ureter by means of a catheter. This was followed by a diminution of pain. Further dilatation was carried out on June 18, 1926, when the catheter passed through a second stricture. Since that time the ureter had taken a full sized catheter.

Dr. Kennedy's last patient was a woman, aged thirty-one years, who had complained of pain over the lumbar spinous processes and in the *erector spinæ* muscles. Tenderness had been present in the right loin along the course of the ureter and tenderness of not so pronounced a degree was also noted on the left side. On cystoscopic examination no abnormality had been discovered in the bladder, but the smallest catheter would not pass further than five centimetres (two inches) up the left ureter. A pyelogram had revealed the presence of a stricture and a dilatation of the ureter. On June 15, 1926, the ureter was dilated and the patient's pain disappeared.

Gastric and Duodenal Ulcer.

Mr. Hugh Trumble, M.C., and Dr. Colin MacDonald demonstrated from a very large number of radiograms secured from various patients prior to and subsequent to operation for gastric or duodenal ulcer.

Genital Prolapse.

Dr. Robert Fowler, Dr. C. S. Wood and Dr. R. D. Aitchison demonstrated from a series of twelve patients who were awaiting operation for the relief of genital prolapse. They further presented eight or nine women cured by purely vaginal operations. Lantern projection was employed by Dr. Fowler to illustrate the steps and technique of these operations.

Dr. Fowler intimated that Fothergill's classification of the varieties and combination of genital prolapse (*prolapsus uteri*) was adopted in the gynæcological clinic at the Alfred Hospital and also his methods of operating. No abdominal operations were performed for the cure of prolapse.

Injury to the Forearm.

Mr. H. C. Colville showed a girl of eight years who had received a severe injury to the right forearm on December 16, 1925, by being dragged for some distance by a motor truck. On the child's admission to hospital it had been found that the forearm was severely lacerated and that most of the skin was missing, the elbow joint was gaping open, the head of the radius being dislocated and projecting on the surface. The whole of the extensor musculature of the forearm between the elbow and the wrist had been torn away so that the radius, ulna and interosseous membrane were exposed throughout their entire length.

Immediate operation had been undertaken, the elbow joint was cleansed, the head of the radius replaced and sutured in position and the soft tissues cleaned and dressed with "Acriflavine."

In the course of prolonged after treatment skin grafting by the Thiersch method had been necessary and had been carried out on January 28, 1926 and again on April 29, 1926, sound healing of the raw surfaces being finally obtained.

At this stage it was noted that although the fingers could be extended by the action of the short muscles of the hand, the gripping power was very poor on account of the complete wrist drop. Accordingly, at operation on July 22, 1926, the tendon of the *flexor carpi ulnaris* muscle had been transplanted to the dorsal aspect of the forearm and attached to the remains of the extensor tendons of the fingers at the level of the wrist joint. As a result of this procedure there was (four weeks later) strong dorsiflexion of the wrist when the fingers were flexed and the hand was capable of a useful grip.

Congenital Dislocation of the Hip.

Mr. Colville also showed three children affected with congenital dislocation of the hip in order to illustrate the different stages in the treatment of this condition. In the case of each of the children reduction had been effected by manipulation under anæsthesia and had been followed by fixation of the limb in plaster of Paris.

The first plaster was applied with the thigh in a position represented by 90° of flexion and 90° of abduction and was allowed to remain for three months; it was then removed and a second plaster applied for a further period of three months, but for the second period the flexion and abduction of the thigh were considerably diminished and combined with a certain degree of internal rotation. For a third period of three months an ordinary plaster "spica" bandage was worn, the thigh being maintained in a position of full extension. Children under treatment for congenital dislocation of the hip were encouraged to walk on the affected limb throughout the whole period.

A number of skiagrams were exhibited to show the relationship of the head of the femur to the acetabulum in the different stages of treatment.

Fractures.

Mr. Colville further showed several children in whom severe fractures in the neighbourhood of the elbow and ankle joints had occasioned immediate gross malposition of the fragments. In each instance it had been found impossible to correct the deformity by manipulation under anæsthesia. All had been treated by open operation and in this manner satisfactory anatomical and functional results had been obtained.

Skiagrams.

Mr. St. Clair Steuart exhibited a radiogram of a hydatid cyst situated in the upper portion of the shaft of the femur. A further radiogram, taken after operation for the extirpation of the cyst seemed to indicate that regeneration of bone had taken place throughout the previously rarefied area.

Mr. Steuart also showed radiograms from a patient who had sustained a fracture dislocation at the hip joint. Reduction by manipulation had been successful although not undertaken until one week after the accident.

Dr. C. E. Dennis exhibited a large number of very interesting radiograms. Included among them was a film which showed subluxation at the *symphysis pubis* and the sacro-iliac joint of the *os innominatum* of the right side.

Another film showed subluxation at the sacro-iliac joint and a dislocation of the opposite hip joint without fracture.

The diagnosis of chronic appendicitis in a child had been made by means of a series of radiograms taken three, five, twenty-four and forty-eight hours after a barium meal. The long dilated appendix was well seen. Subsequent operation confirmed the diagnosis.

Asthma.

Dr. C. Sutherland demonstrated from a series of patients, all of whom were subjects of asthma. He performed various cutaneous tests and discussed in detail the technique of desensitization.

Dermatitis Herpetiformis.

Dr. S. W. Shields showed a female, aged sixty-three years, who for the previous six years had suffered from typical grouped erythematous vesicular lesions which were

most pronounced over the scapular region. The lesions had become more or less generalized over the trunk and limbs and were accompanied by intense irritation. The general health was good.

Bazin's Disease.

Dr. Shields also showed a woman, aged twenty-three years, on the calves of whose legs were seen bilateral scars of old ulcers with fresh purplish-red indurated plaques. The patient gave a history of having suffered from tuberculous adenitis in the neck. She had come from England seven years previously.

Darier's Disease.

Dr. Shields's third patient was a woman, aged forty-seven years, whose general health was good. The patient was a grandmother. The lesion had first started as a dirty-looking roughness of the skin of the arms when she was fifteen years of age. The lesions became more or less generalized, but had improved gradually with hot baths and strong salicylic applications. The arms and clavicular regions were most affected and typical crusted papules were present.

Mucous Adenomata.

The last patient shown by Dr. Shields was a man, aged forty-three years, who was suffering from mucous adenomata of the roof of the mouth. The lesion consisted of small protuberant spots of yellowish colour occurring in crops.

Post-Graduate Work.

THE ANNUAL POST-GRADUATE COURSE IN MELBOURNE.

The Melbourne Permanent Committee for Post-Graduate Work have issued the syllabus for the annual post-graduate course which will be held from November 8 to November 20, 1927. The fee for the course is three guineas, payable at the time of entry or before the commencement of the course. All applications should be sent to the Honorary Secretaries, 12, Collins Street, Melbourne, not later than October 31, 1926. Applicants are requested to intimate at the time of joining their address in Melbourne during the period of the course. The central information office will be situated at the Walter and Eliza Hall Institute for Research in Pathology and Medicine, Melbourne Hospital.

Syllabus.

Monday, November 8, 1926.

11.15 a.m.—Dr. Hume Turnbull: "The Causes of Heart Failure," at the Melbourne Hospital.
Mr. Alan Newton: Demonstrations in the surgical wards at the Melbourne Hospital.
2.15 p.m.—Dr. C. H. Kellaway: "Focal Infections," at the Melbourne Hospital.
3.30 p.m.—Professor P. MacCallum: Pathological Demonstrations at the Melbourne Hospital.
8.30 p.m.—Inaugural Address by Sir George Syme at the Medical Society Hall, East Melbourne.

Tuesday, November 9, 1926.

9.30 a.m.—Mr. H. B. Devine: "Atypical Forms of Appendicitis," at the Saint Vincent's Hospital.
Dr. A. J. Brennan: "Laboratory Methods for General Practitioners," at the Saint Vincent's Hospital.
11.15 a.m.—Dr. L. S. Latham: Demonstrations of patients with pulmonary diseases, at the Saint Vincent's Hospital.
Mr. J. Newman Morris: "Surgery of the Hand and Wrist," at the Saint Vincent's Hospital.
2.15 p.m.—Dr. L. J. Clendinnen: "Radiography," at the Melbourne Hospital.
3.30 p.m.—Dr. Leonard Mitchell: "Eye Cases of Everyday Practice," at the Melbourne Hospital.

Wednesday, November 10, 1926.

9.30 a.m.—Dr. J. S. Green: "*Post Partum* Hæmorrhage: The Use and Abuse of Pituitrin," at the Women's Hospital.
11.15 a.m.—Dr. W. Ivon Hayes: "Narcosis in Childbirth," at the Women's Hospital.
2.15 p.m.—Dr. H. M. Hewlett: "Gastro-Intestinal Radioscopy," at the Saint Vincent's Hospital.
3.30 p.m.—Dr. L. Doyle: "Local Anæsthesia," at the Saint Vincent's Hospital.
8.15 p.m.—Professor R. J. A. Berry: Demonstrations in anatomy at the Anatomy School, University of Melbourne.

Thursday, November 11, 1926.

9.30 a.m.—Dr. Julian A. R. Smith: Operations with demonstrations arranged with a view to utility in general practice, at the Saint Vincent's Hospital.
Dr. J. W. Grieve: "Acute Infections of the Central Nervous System," at the Saint Vincent's Hospital.
11.15 a.m.—Mr. C. Gordon Shaw: "The Diagnosis of Lesions of the Right Iliac Fossa," at the Saint Vincent's Hospital.
Dr. F. Apperly: "Some Types of Dyspepsia," at the Saint Vincent's Hospital.
2.15 p.m.—Dr. A. H. Thwaites: "Radiotherapy," at the Alfred Hospital.
3.30 p.m.—Mr. H. C. Trumble: "Diathermy," at the Alfred Hospital.

Friday, November 12, 1926.

9.30 a.m.—Dr. S. V. Sewell: "Coma," at the Melbourne Hospital.
Mr. W. A. Hailes: "Tumours of the Bladder," at the Melbourne Hospital.
11.15 a.m.—Mr. V. Hurley: Demonstrations in the septic wards, at the Melbourne Hospital.
Dr. Douglas J. Thomas: "Dietetic Management of Diabetics," at the Melbourne Hospital.
2.15 p.m.—Dr. S. W. Ferguson: "Infant Feeding Principles," at the Children's Hospital.
3.30 p.m.—Dr. J. G. Whitaker: Demonstrations in the surgical wards, at the Children's Hospital.
7.30 p.m.—Dr. Kenneth A. McLean: "Venereal Diseases," at the Melbourne Hospital.

Saturday, November 13, 1926.

9.30 a.m.—Mr. Harold R. Dew: "Swellings of the Lymphatic Glands," at the Melbourne Hospital.
Dr. L. E. Hurley: "Auricular Fibrillation," at the Melbourne Hospital.
11.15 a.m.—Dr. R. P. McMeekin: "Neurology in General Practice," at the Melbourne Hospital.
Dr. C. W. B. Littlejohn: "Pain in the Back," at the Melbourne Hospital.

Monday, November 15, 1926.

9.30 a.m.—Dr. A. V. M. Anderson: "The Diagnosis and Treatment of Nephritis," at the Alfred Hospital.
Mr. A. J. Trinca: "The Early Diagnosis of Cancer of the Breast," at the Alfred Hospital.
11.15 a.m.—Dr. R. St. C. Steuart: "Common Knee Joint Injuries," at the Alfred Hospital.
Dr. J. P. Major: "Type of High Blood Pressures," at the Alfred Hospital.
2.15 p.m.—Dr. H. Laurie, Dr. H. C. Colville and Dr. F. K. Norris: Demonstrations on children's diseases, at the Alfred Hospital.

Tuesday, November 16, 1926.

9.30 a.m.—Mr. J. T. Tait: "Urethral Stricture," at the Melbourne Hospital.
Dr. W. W. S. Johnston: "Uræmia," at the Melbourne Hospital.
11.15 a.m.—Mr. T. E. L. Lambert: "Head Injuries," at the Melbourne Hospital.
Dr. F. B. Lawton: "Cardio-Vascular Syphilis," at the Melbourne Hospital.
2.15 p.m.—Dr. R. R. Wettenhall: "Common Skin Ailments," at the Melbourne Hospital.

Wednesday, November 17, 1926.

9.30 a.m.—Dr. A. Sherwin: "Indications for Operation in Gynæcology," at the Women's Hospital.
11.15.—Dr. R. N. Wawn: "Indications for Interference in Labour," at the Women's Hospital.
2.15 p.m.—Dr. F. G. Morgan: Demonstrations of serum, vaccines, at the Commonwealth Serum Laboratories.
Dr. A. J. W. Philpott and Dr. Dr. W. de Witt Henty: "Mental Diseases Met With in General Practice," at the Acute Mental Hospital.
8.15 p.m.—Dr. R. Marshall Allan: "Obstetrical Practice," at the Medical Society Hall, East Melbourne.

Thursday, November 18, 1926.

9.30 a.m.—Mr. B. Kilvington: "Fractures of the Femur," at the Melbourne Hospital.
Dr. K. Hiller: Demonstrations in the surgical wards of the Melbourne Hospital.
11.15 a.m.—Mr. B. T. Zwar: "The Diagnosis of Upper Abdominal Lesions," at the Melbourne Hospital.
Dr. S. O. Cowen: "Aortic Diseases," at the Melbourne Hospital.
2.15 p.m.—Dr. F. V. Scholes: "Infectious Diseases," at the Queen Victoria Hospital for Infectious Diseases.
Dr. K. G. Colquhoun: Demonstrations in the dermatological wards, at the Saint Vincent's Hospital.
8.15 p.m.—Professor F. Wood Jones: "The Central Nervous System," at the Medical Society Hall, East Melbourne.

Friday, November 19, 1926.

9.30 a.m.—Mr. Balcombe Quick: "Surgery Abroad," at the Alfred Hospital.
Dr. R. J. Bell: "Insulin and How to Use it," at the Alfred Hospital.
11.15 a.m.—Dr. J. F. Mackeddie: "Artificial Pneumothorax," at the Alfred Hospital.
Mr. J. Kennedy: Demonstrations on fractures, at the Alfred Hospital.
2.15 p.m.—Dr. R. M. Downes: "Heliotherapy," at the Brighton Convalescent Home.
Dr. J. B. D. Galbraith: "Pulmonary Diseases in Children," at the Children's Hospital.
8.15 p.m.—Professor F. Wood Jones: "The Central Nervous System," at the Medical Society Hall, East Melbourne.

Obituary.

GEORGE JOSEPH CHIPPERFIELD.

The residents of Canowindra, New South Wales, and the surrounding district suffered a grievous loss by the death of Dr. George Joseph Chipperfield which occurred at Canowindra on August 11, 1926.

Born in London on September 15, 1869, George Joseph Chipperfield studied medicine in Canada. He took the degrees of doctor of medicine and master of surgery at the University of Manitoba. On graduation he practised for a time at Brandon, in Winnipeg, and then proceeded to Edinburgh where in 1898 he became licentiate of the Royal College of Physicians and the Royal College of Surgeons of that city. He also was admitted as licentiate to the Fellowship of Physicians and Surgeons of Glasgow. He then returned to Canada and practised for a while at Revelstoke, British Columbia. In 1905 he came to Australia and practised at Gunnedah, Gilgandra, Cambellego and finally at Canowindra. At the latter town he held the position of Government Medical Officer and was also a medical officer of the Soldiers' Memorial Hospital. In 1921 he went for a health trip to Europe and Canada. He did not enjoy good health, however, after his return and one month before his death suffered from an attack of influenza. He was not able to combat the infection successfully and suffered from a relapse. To this he finally succumbed.

George Joseph Chipperfield served his town and district faithfully and well. He was beloved of his patients who gave striking evidence after his death of their regard for him. He is survived by a widow and two daughters.

ALEXANDER MORRISON.

We regret to announce the death of Dr. Alexander Morrison which occurred at Ipswich, Queensland, on September 29, 1926.

ANDREW HONMAN.

We announce with regret the death of Dr. Andrew Honman which occurred at Melbourne on October 3, 1926.

UNIVERSITY OF SYDNEY CANCER RESEARCH FUND.

We have pleasure in announcing that Dr. E. Danziger, of Port Melbourne, has forwarded to us one pound in response to our appeal on behalf of the University of Sydney Cancer Research Fund. Books of stamps can be had on application to the office of the journal. Each book contains twenty stamps and the cost is one pound.

Correspondence.

OCCIPITO-POSTERIOR POSITIONS.

Sir: I quoted Williams as being in favour of rotation with forceps and I admit, as Dr. Gibson states, that he tries manual rotation first, but when that fails he rotates with forceps. He speaks of this method as one "which has given such excellent results in my hands that I employ it to the exclusion of all other methods," that is after failure with manual rotation. Further on, however, he says: "By this method I have obtained most satisfactory results and have been able to deliver many women with ease when the usual methods have failed. Indeed my experience has been so satisfactory that I have ceased to dread occipito-posterior positions and now regard them with equanimity, feeling that delivery can be safely and readily effected when necessary."

My view on reading this enthusiastic eulogy of the method was that if delivery can thus be safely and readily effected when the usual methods have failed, why should it not be tried first?

I have found that rotation can be more easily accomplished by first pushing the head up to the pelvic brim, as advised by Dr. Gibson, in manual rotation and I do not think that this departure from Williams's description has added danger to the method, for experience teaches us that the less force one uses, the less trauma results.

With regard to morbidity, I can only repeat that I have not seen any damage to mother and child with this method, but I know that considerable damage can be caused to both when forceps extraction in the occipito-posterior position is carried out. Yet this is advised by many authorities when manual rotation fails. In many cases the child cannot be delivered in this way and it is surely one of the commonest causes of still-birth and maternal invalidism. I do not deny that morbidity may occur after rotation with forceps, as after any manipulative procedure, but manual rotation also has its share in this respect as well as its percentage of failures.

Dr. Gibson mentions the numerous cases of scar tissue in the vaginal vault after forceps delivery, but does not state whether any were attributable to rotation with forceps. Such information would be difficult to obtain in the out-patient department of a metropolitan hospital where patients come from various districts, but in many of the cases would be due to forceps extraction in cases of contracted pelvis or postmature babies. Certainly a fairly large proportion would be due to extraction in the occipito-posterior position and some (one regrets to say) to hasty delivery through a partially dilated cervix.

One must pay attention to the statistics published in the *American Journal of Obstetrics and Gynecology* showing a morbidity of 30% with the Scanzoni method, though statistics are not always reliable, but the experience of

one's fellow practitioners is also of considerable importance and several men of many years' general practice and wide obstetric experience have told me that they have found this method of the greatest value and have no intention of abandoning it. They had not found that it caused the damage it was said to do.

All will agree with Dr. Gibson's statement that the posterior position should be recognized before the thirty-sixth week and efforts made to convert it into an anterior one—the importance of antenatal supervision cannot be over-emphasized, but in my letter I discussed only the stage where labour had been in progress some time and the head discovered in a posterior position.

The treatment of occipito-posterior positions has not been standardized and one uses the method he finds best and I am not alone in my preference for the one I have described. It has stood to me so often in the past and has proved so safe that I felt bound to protest against its condemnation as not being quite justified.

Yours, etc.,

JAMES C. HUGHES, M.B., Ch.M.

Lang Road, Centennial Park,
Sydney, September 22, 1926.

COMPENSATION AND INJURY TO THE BACK.

SIR: Referring to my letter under date July 20 dealing with compensation and injury to the back, your correspondents seem to have missed the point I raised. I made no mention of malingering and referred to genuine cases of disability occurring suddenly when at work and ascribed by the patient and often by his doctor also to strain, wrench or tearing of lumbar tissues. I do not include cases in which there has occurred interruption of neuromuscular coordination; I include only those in which an accustomed simple act is performed. Let us suppose two men stoop, one picks up a postage stamp, the other a shovel. Both are suddenly seized with the condition generally known as "lumbago." Are they traumatic? If both, why? What is the difference in the affected tissues since the stamp or the shovel was lifted? And why is that particular tissue affected and not others synchronously used? If the latter case only is accidental what is the former? How can I tell apart from the patient's tale between the traumatic and non-traumatic?

My primary object in raising the question is to endorse or correct my opinion that accustomed use only of any tissue cannot produce sudden disability of those tissues. If I am right, the man who ascribes his lumbar disability to lifting a shovel, tipping a draw, tying a boot lace *et cetera* is wrong. Therefore he has not shown that such disability has more to do with or aggravated by the nature of his employment than with that of his amusements. He has merely stated that he was at work when his attention was drawn to his condition.

Yours, etc.,

GERALD S. SAMUELSON.

Lane Cove,
September 30, 1926.

SIR: In reply to Dr. Hoets's criticism of my letter: I am glad to get his views as to the true nature of the condition of "strained back." In regard to the first point: What is true rheumatism of the lumbar muscles? I admit it is a difficult question and there are many views concerning its nature. I should construe it as an inflammation of the muscles, their nerves and nerve terminations brought on by cold or overstrain or both combined. One frequently meets with this condition in a man who has been doing some strenuous work such as heavy lifting and has strained or overstretched his muscles. As long as he is working and keeping warm, he does not feel any inconvenience, but when he ceases work for a while, especially if he has been perspiring freely, he finds when he starts work he is stiff and movements of the muscles cause pain. In this case the muscles from being overstretched are injured temporarily and are especially liable in a rheumatic subject or one addicted to alcohol to be affected adversely by cold or damp. This condition contrasts very strongly with the case of one who, while lifting a heavy weight, suddenly feels a sharp pain the severity of which compels him to cease work at once. In this case one would suspect a rupture of some fibrous structure especially at its insertion into the bone and the periosteum injured as well. The pain here would be constant from the start and the case would require a much longer period of rest than in the case of simple overstretched muscles.

In regard to my statement of the back being bent at the "lumbo-sacral" joint I admit the slip; it should have read "hip" joint and I am glad Dr. Hoets has called attention to it. His explanation of the flexed position is interesting, though I am inclined to think the position of greatest ease is the predominant factor in assuming the attitude of flexion, the altered curvature of the lumbar spine following as a natural sequence in order to balance the shifted centre of gravity. I find it a little difficult to conceive of an inflamed or strained muscle, where this is involved, being in a state of contraction in fixing the lumbar vertebræ, as Dr. Hoets asserts, without causing severe pain, yet the fact remains that the flexed position by relaxing the muscles and ligaments is the position of greatest ease.

In regard to X ray examination for diagnosis I can quite understand its value in determining whether there is detachment of fibres, tendons or a spicule of bone, but I fail to see clearly any great value in determining whether the condition is one of strained or inflamed fibrous or muscular tissue or a case of malingering except in so far as the malingerer, being in strange surroundings, his attention might be deflected sufficiently to put him off his guard and unmask him as Dr. Hoets points out.

Yours, etc.,

E. C. CHISHOLM.

Comboyne,
September 20, 1926.

ENUCLEATION OF TONSILS.

SIR: The correspondence on the question of the enucleation of tonsils is again forcing these evergreen (or should one say ever-red) offenders into the open to meet the disapproving gaze of most men of medicine who are given to thinking how best to render them fit company for the other members of the body physical or failing that how best to remove them.

Like Dr. Baxter I feel that for the present the only safe course is to remove them. The surgical procedures at present in vogue are almost as various as their users and anything approaching a standardization of methods is to be welcomed. Such standard procedure must be mechanically and anatomically sound, must be practicable in the hands of average surgical skill under reasonable conditions of modern operative procedure and must be applicable in 100% of cases. Guillotine advocates, while having much in their favour, are obliged to acknowledge the failure of their method in at least 10% of all cases (and that is allowing for great enthusiasm and skill).

The protagonists of dissection find some form of insufflation anæsthesia necessary and this is largely intratracheal anæsthesia. This group I imagine would hesitate to recommend this course in young children as a routine even where apparatus is available in competent hands. Local anæsthesia is of course not here considered owing to the limited field of its usefulness for this operation.

In your last issue Mr. Kent Hughes writing on the subject makes some assertions that are hard to accept and I feel sure that he would do us a service if he were to be more explicit. With his extensive experience in this field he doubtless has good reasons for adopting his own procedure and has detected the fallacy in other methods. Tonsillectomy is more than 50% a question of mechanics and it seems to me that Dr. Hennessy's contribution is a great aid in the standardization of this part. Now would Mr. Hughes tell us whether it is the whole or part that he condemns and, if the latter, explain where and why.

Despite his fatality with the head fully extended, does he not think that it is mechanically correct where blood is escaping from a raw tonsil bed surface and the chief concern is to keep it out of the lower air passages? Also while avoiding special instruments by which I take it he means chiefly the gag, in what class does he place a guillotine, long scissors curved on the flat and Mackenzie's tonsil dissector? As he is frankly opposed to a procedure that he says he has abandoned, his reasons for avoiding the separate steps would be helpful to others of us to see the weakness where we fail to detect any at present.

Yours, etc.,

Arthur Murphy,
Honorary Ear, Nose and Throat Surgeon,
Children's Hospital, Brisbane.

Brisbane,
September 28, 1926.

CONGENITAL INSANITY AND DEFECTIVE DEVELOPMENT.

Sir: Dr. Clif Tucker's instructive paper, "Points from Practice: Obstetrical" in the issue of September 25 calls to mind a thought which has often cropped up in regard to the treatment of threatened abortion. I write this seeking information and not in a critical sense. Is an embryo permanently damaged in any way by having its nidus disturbed by a threatened abortion? I know of two cases, one of which was followed by idiocy and the other by a double cleft palate and harelip and my personal feeling has always been that I would rather let that particular pregnancy terminate itself than try and patch it up. The case notes of those who do a big midwifery practice should clear up this question. There are certain types of congenital insanity where defective development of the embryo is suspected as the cause, hence my interest in the subject.

Yours, etc.,

W. A. T. Lind,
Victorian Lunacy Department.

Kew, Victoria,
September 30, 1926.

GALL BLADDER AND ALLIED INFECTIONS.

Sir: Repeated perusal of Dr. H. Skipton Stacey's thoughtful article (The Medical Journal of Australia, August 28, 1926) has emphasized certain questions long in the writer's mind as to the after-results of cholecystectomy.

The textbooks and many articles in the literature would lead one to infer or to believe that the removal of an infected gall bladder nearly always results in a cure of the infection. Observation has convinced one that this is not the case. How can it be if "in many cases hepatitis is a precedent condition to the cholecystitis," *id est* "infection in these cases is presumably downward from the liver by the bile ducts . . . to the gall bladder"? How can it be in the light of Graham's findings? One knows of cholecystectomized patients (without stones in the ducts) who still—in spite of a strict cholesterol-free diet—suffer from "shivery feelings" down the back and from "bilious attacks" (with or without jaundice) at times distinctly excited by exposure to cold, and who give evidences in other ways of diminished hepatic efficiency and of residual infection. In other words there is still present a cholangitis of varying degree.

The gall bladder cannot be regarded as "walled off" from the remainder of the biliary system—"it has now been shown that inflammation of the gall bladder is accompanied by hepatitis" (Rolleston).

Therefore theory and practice lead one to the conclusion that drainage of the common bile duct should always follow cholecystectomy done for infection. This is not taught in the books, if at all. Moynihan, for example, in "Abdominal Operations" stresses the importance of drainage after the removal of calculi from the ducts, but makes it plain that he does not practise direct drainage in cholecystectomy.

Possibly the routine drainage of the common duct might cause the "in most cases" of Dr. Stacey's fourth conclusion to be changed to "in all cases."

With regard to after treatment how many men realize the necessity for a cholesterol-free diet, perhaps permanently?

The writer would greatly like to see a free expression of opinion in the correspondence columns of the journal on the following questions, particularly by men who have been able to observe their cases for some years after operation. (In that connexion I am sure that Dr. Stacey will agree that it is yet too early to claim cures in the two cases of frontal pain cited by him or even to determine the cause of its disappearance.)

Questions:

1. Is it right to conclude that direct drainage of the common bile duct should form part of the operation of cholecystectomy in cases of infection?
2. What is the best technique for drainage of the common bile duct?
3. In early cases of gall bladder and hepatic infection without calculi is it good practice to do a cholecystostomy and thus give the biliary system a chance to recover?
4. In early cases will cholecystectomy alone clear up the infection and if in any given case it does not, how much worse off is the patient?

Yours, etc.,

F.R.C.S.

September 28, 1926.

POST-GRADUATE HOSTEL.

Sir: May I draw attention to several points of interest concerning our hostel. It presents all the advantages of a club with few of its disadvantages.

1. No introduction beyond the presentation of a visiting card is required. This is of importance for dominion men and women who arrive here unacquainted.
2. There is no entrance fee. This is a consideration for anyone spending perhaps only a week or two in London every few years and to those who can "drop in" only very occasionally in the evening.
3. A doctor may bring his wife and family and whilst not enjoying the privileges of the rooms reserved for members they can at least be under the same roof.
4. Women practitioners may have access to all the discussions and rooms except the smoking-room.
5. A ladies' subcommittee will be formed at an early date to care for the interest of the family of a medical man as it is realized what sacrifices they make when a doctor takes a "busman's holiday."

Yours, etc.,

A. P. Bertwistle.

Post-Graduate Hostel,
Imperial Hotel,
Russell Square, London, W.C.1,
August 27, 1926.

Proceedings of the Australian Medical Boards.

TASMANIA.

The undermentioned have been registered, under the provisions of *The Medical Act* 1918, as duly qualified medical practitioners:

Baker, Bernard Allen, M.B., B.S., 1925 (Univ. Melbourne), Wynyard.
Butchart, Russell Exon, M.B., B.S., 1920 (Univ. Melbourne), F.R.C.S., 1925 (Edin.), Waratah.
Farrell, William George, M.B., B.S., 1926 (Univ. Melbourne), Catamaran.
Maplestone, Philip Alan, M.B., B.S., 1908 (Univ. Melbourne), St. Helens.

Books Received.

ADVICE TO THE EXPECTANT MOTHER ON THE CARE OF HER HEALTH, by F. J. Browne, M.D., D.Sc., F.R.C.S.E.; 1926. Edinburgh: E. & S. Livingstone. Crown 8vo., pp. 40. Price: 6d. net.

HANDBOOK OF MEDICAL ELECTRICITY AND RADIOLOGY, by James Riddell, F.R.F.P.S.; 1926. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 254, illustrated. Price: 8s. 6d. net.

ORTHOPÆDIC SURGERY, by W. A. Cochrane, M.B., Ch.B., F.R.C.S.E.; Edinburgh: E. and S. Livingstone. Demy 8vo., pp. 551, illustrated. Price: 21s. net.

CARBOHYDRATE METABOLISM AND INSULIN, by John James Rickard Macleod, F.R.S., M.B., LL.D. (Abdn.), D.Sc. (Hon.) (Toronto); 1926. London: Longmans, Green and Company, Limited. Royal 8vo., pp. 369, with illustrations. Price: 18s. net.

EARLY MENTAL DISEASE, by a Group of Well-Known Authorities; *The Lancet* Extra Numbers No. 2; 1926. London: Wakley and Son (1912) Limited. Sydney: Angus and Robertson Limited. Imp. 8vo., pp. 200. Price: 12s. net.

THE LAW AND PRACTICE RELATING TO DENTISTS AND DENTISTRY, by Albert Crew; 1926. London: John Bale, Sons and Danielsson, Limited. Crown 8vo., pp. 244. Price: 10s. 6d. net.

ANNALS OF THE PICKETT-THOMSON RESEARCH LABORATORY, Volume II.; 1926. London: Baillière, Tindall and Cox. Demy 4to., pp. 175, with illustrations. Price: 42s. net.

AIDS TO CASE-TAKING, by Henry Lawrence McKisach, M.D., F.R.C.P. (London); Second Edition; 1926. London: Baillière, Tindall and Cox. Foolscap 8vo., pp. 175. Price: 4s. 6d. net.

HYDROGEN ION CONCENTRATION OF THE BLOOD IN HEALTH AND DISEASE, by J. Harold Austin and Glenn E. Cullen; 1926. Baltimore: The Williams and Wilkins Company. Royal 8vo., pp. 86, with illustrations. Price: $2.00 net.

Medical Appointments.

Dr. Arthur Norris Wilkinson (B.M.A.) has been appointed Public Vaccinator at Yea, Victoria.

* * * *

Dr. John Francis Gaha (B.M.A.) has been appointed a Member of the Council of the University of Tasmania.

* * * *

Dr. Joshua Law Kerr has been appointed a Justice of the Peace for the District of Esperance, Tasmania.

* * * *

Dr. J. McAleer (B.M.A.) has been appointed Quarantine Officer, Geraldton, Western Australia.

* * * *

Dr. Henry Rogerson (B.M.A.) has been appointed Acting Medical Superintendent of the Hospital for the Insane, Kew, Victoria.

* * * *

Dr. H. R. G. Poate (B.M.A.) has been appointed Acting Assistant Commissioner-in-Charge of the New South Wales District of the St. John Ambulance Brigade in the place of Dr. T. Morgan Martin (B.M.A.) who recently resigned.

* * * *

Dr. Clarence Michael Guiney (B.M.A.) has been appointed Government Medical Officer at Bourke, New South Wales.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants *locum tenencies* sought, etc., see "Advertiser," page xx.

Isisford District Hospital, Queensland: Medical Officer.
Mount Mulligan District Hospital: Medical Officer.
Victorian Baby Health Centres Association, Melbourne: Medical Officer.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
New South Wales: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
Queensland: Honorary Secretary B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
South Australian: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Ceduna, Wudinna (Central Eyre's Peninsula), Murat Bay and other West Coast of South Australia Districts.
Western Australian: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
New Zealand (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

Oct. 18.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Oct. 19.—Tasmanian Branch, B.M.A.: Council.
Oct. 19.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Oct. 20.—Western Australian Branch, B.M.A.: Branch.
Oct. 20.—Central Northern Medical Association, New South Wales.
Oct. 21.—Section of Neurology and Psychiatry, New South Wales Branch, B.M.A.
Oct. 22.—Queensland Branch, B.M.A.: Council.
Oct. 23.—Illawarra Suburbs Medical Association, New South Wales.
Oct. 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Oct. 27.—Victorian Branch, B.M.A.: Council.
Oct. 28.—New South Wales Branch, B.M.A.: Branch (Ordinary).
Oct. 28.—South Australian Branch, B.M.A.: Branch.
Nov. 2.—Tasmanian Branch, B.M.A.: Council.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

All communications should be addressed to "The Editor," The Medical Journal of Australia, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

Subscription Rates.—Medical students and others not receiving The Medical Journal of Australia in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.